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ROKITANSKY'S

PATHOLOGICAL

ANATOMY.

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SYDENHAM SOCIETY

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LONDON

MDCCCLIV.

A MANUAL

OF

PATHOLOGICAL ANATOMY.

BY

CARL ROKITANSKY, M.D.

CURATOR OF THE IMPERIAL PATHOLOGICAL MUSEUM, AND
PROFESSOR AT THE UNIVERSITY OF VIENNA, ETC.

IN FOUR VOLUMES.

VOL. I.

LONDON:
PRINTED FOR THE SYDENHAM SOCIETY.
MDCCCLIV.

PRINTED BY J. E. ADLARD,
BARTHOLOMEW CLOSE.

A MANUAL

OF

GENERAL PATHOLOGICAL ANATOMY.

TRANSLATED FROM THE GERMAN

BY

WILLIAM EDWARD SWAINE, M.D.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;
PHYSICIAN EXTRAORDINARY TO H. R. H. THE DUCHESS OF KENT.

EDITOR'S PREFACE

TO VOL. I.

In issuing this portion of Rokitansky's 'Pathological Anatomy,' it is necessary to offer, on behalf of the Council of the Sydenham Society, some apology for the delay which has attended the completion of this important and voluminous work. In his interesting preface to the second volume, Dr. Sieveking has recorded *one* reason for the order in which the volumes have been published; but he has not adverted to the main consideration by which the Council was influenced, namely, the apparently well-founded hope that they might be enabled to present the Association with the histological portion of the work *in a new and revised edition.* Encouraged from time to time in this hope by the Author himself, the Council did not hesitate to defer, from year to year, the publication of the first volume, until they felt that it would be improper to tax the patience of the members any further. The new edition is still promised, but with no surer pledge for its early completion than heretofore! The Editor has, however, availed himself to a considerable extent of certain papers read by the Author before the Imperial Academy of Sciences at Vienna; namely, On the Structure and Growth of Cyst and of Cancers, &c. He has even found it not at all incompatible with the general unity and concordance of the work to substitute, almost bodily, the

Author's more recent essay on 'Cyst and Alveolus,' for the comparatively brief and imperfect article on the same subject in the original. These papers, there is reason to believe, contain the principal results of the Author's more recent investigations, and therefore, in all probability, the most important of the additions that might be anticipated in a new edition. The Council has also sanctioned the introduction of two plates in illustration of the newly added matter.

At the conclusion of the present volume will be found a copious index to the four volumes collectively. To this each Editor has contributed his respective share, thus offering to the English reader facilities altogether wanting in the original work.

On the other hand, the Editor has felt the necessity of abridging somewhat the Author's general introduction, partly because, totally unlike the general tendency of the work, it is of too "transcendental" a character either to suit the English language or to harmonize with English ideas; but more particularly because it is interwoven with a train of speculative reasoning upon the relation between power and matter, which might, in this country, very possibly give rise to misinterpretation and rebuke.

What Dr. Sieveking justly alleges of the general peculiarities of Rokitansky's style, and of the difficulty of rendering his writings intelligible in English, is, by all who are conversant with the original, admitted to apply with especial force to the first volume. Upon this ground the Editor ventures to urge his claim for a fair measure of indulgence on the reader's part.

In conclusion, the Editor, having been disappointed of a promised auto-biographical sketch, takes leave to subjoin a few extracts from a short account of the career of this great pathologist, copied by a friendly hand from the last edition [1854] of Brockhaus's 'Conversation's Lexicon.'

"Charles Rokitansky, the founder of the German [it should rather have been called, Austrian] medico-anatomical school, was born at Königsgraetz, in Bohemia, was educated at the Gymnasium of Leitneritz, and graduated, at Vienna, in 1828. Shortly afterwards he was appointed Assistant in the pathologico-anatomical department of the University, and, in 1834, Professor of Pathological Anatomy. At the same time he was instituted Prosector at the General [united Civil and Military] Hospital at Vienna, and also sole medico-legal Anatomist for the examination of all doubtful cases of death throughout that metropolis.

"The immense fund of materials thus placed at his disposal [the number of corpses dissected by him is summed up at 30,000] was almost entirely reserved for the elaboration of that grand work on pathological anatomy, which, in the consciousness of having thoroughly mastered the subject, he gave to the world between the years 1842 and 1846; which has passed, *unaltered,* through three reimpressions; and which, under the auspices of the Sydenham Society, has been translated into the English language."

"In 1849 Rokitansky was appointed Dean of the Medical Faculty, and, in 1850, Rector of the University, of Vienna."

YORK; *January*, 1855.

AUTHOR'S PREFACE.

The appearance of this first volume brings the publication of my 'Pathological Anatomy' to a close. As was the case with the earlier volumes, the completion of this one has been delayed by lack of leisure, and especially by long and repeated attacks of illness.

Whilst engaged in working out the design of this Pathological Anatomy, I have throughout endeavoured to act the part of a clinical teacher; and I believe that, in so doing, I have apprehended the requirements of our day, and usefully disposed of the colossal materials within my reach.

The same self-reliance that characterized the commencement of my pathologico-anatomical studies, has stood by me whilst engaged in observing and interpreting the facts of which the said materials are composed: for, each individual discovery encouraged me more and more to pin my faith upon Nature alone. Still I have never failed to watch and to appreciate the achievements of other men.

The present work will at any rate tend to show, how thorough is my conviction that Pathological Anatomy must constitute the groundwork, not alone of all medical knowledge, but also of all medical treatment; nay, that it embraces all that medicine has to offer of positive knowledge, or at least of what is fundamental to it. Its domain will here, however, be found more extended, and more nearly approximated to the confines of pathological Chemistry than has generally been the case in pathologico-anatomical writings.

Upon individual sections of the work I must confess to have exercised a certain favoritism; and I have striven to cultivate and to carry out some important general views, with a well-tested conviction of their truth. Amongst these views I may here single out for exemplification the doctrine of a primitive diversity in blastemata, as the only tenable basis for a humoral pathology.

From a comparison of the antecedently published volumes on special pathological anatomy with the present one, it will be seen that the former furnish the groundwork of the views here propounded, and that my convictions, upon the whole, remain unchanged.

THE AUTHOR.

VIENNA; *July*, 1846.

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INTRODUCTION.

Pathological Anatomy may be said to be a modern science. It is indeed only of late years that it has assumed the dignity of an independent science at all.

Although, according to Pliny, dead bodies were examined in Egypt at the time of the Pharaohs, that is to say, many centuries before Galen, with a view to detect the seats of disease; the result of those researches has remained unrevealed to us. Even upon Greek medicine the pathologico-anatomical observations made by its founders and scholars have been without material influence. They were indeed gradually lost sight of in the medical schools which arose out of the successive systems of philosophy of a later period.

Not until the commencement of the sixteenth century—the period of the regeneration of anatomy—does the epoch begin of an occasional, fragmentary, indeterminate study of pathological anatomy. Still, Eustachius, the rival of Vesalius, must have been deeply impressed with its importance; for, towards the close of his life he expresses his regret that he had not rather bestowed upon pathological anatomy that time and attention which he had devoted to physiological anatomy. The first who dedicated himself in an especial manner to pathological anatomy was Antony Benivieni, who wrote, at Florence, 'De abditis morborum causis' (1507). He was followed by Mathieu-Reald Columbus, the protector of Vesalius (1590), Volcher Coiter, a disciple of Fallopius (1573), Salius Diversus

(1584), Marcellus Donatus (1588). Johannes Schenkins collected the observations made up to his time (1584). Johannes Wierus (1569), Felix Plater (1614), Fabricius Hildanus (1606), Tulpius (1672), Vesling (1664), Thomas Bartholin (1654—1675), Stalpaart van der Wiel (1677), Daniel Sennert (1676), Friedrich Ruysch (1691), cultivated pathological anatomy after their own fashion. Their observations, although partially of great interest, often bear the impress of superstition, and are disfigured by the fanciful way in which they are interpreted.

Since the time of Harvey, the discoverer of the circulation, who, in denominating our particular science, *medical anatomy*, showed how fully he comprehended its import, various physicians have worked out sundry branches of pathology anatomically. Amongst them are Thomas Willis (1677) and J. J. Wepfer (1658—1727). Others, as Fernel (1679), F. Sylvius (1734), Baillou (1735), have, in their compendia of pathology, adopted pathological anatomy for their groundwork. Bonnet was, however, the first who compiled an ample repertory on this subject ('Sepulchretum,' 1679); and even this work unites to the imperfections of earlier observations the lack of a standard physiological principle, and of a definite practical tendency. The same applies equally, if not more forcibly, to Blankaard's 'Anatomia practica' (1688).

Above both these—above all that had been previously accomplished—stand pre-eminent, Morgagni and his work, 'De sedibus et causis morborum' (1767). Notwithstanding its defects, this book remains a model of industry and perseverance, of method and arrangement, of breadth and perspicuity, and, lastly, of originality, for all time.

In the same century, special investigations, not unworthy of record, were made by J. Moritz Hofman, Walter, Albinus, Vater, Levret, W. Hunter, Senac, Meckel, Böhmer, Van Doeweren, Camper, Bleuland, and others.

In a work containing a vast number of facts ('Historia anatomico-medica,' 1768), the purpose attained by Morgagni, failed in Lieutaud's hands, through lack of detail, of analysis, of a practical generalisation of facts. On the other hand, Sandifort ('Observ. anat. path.,' 1777) merits, for the richness and solidity of his writings, to be classed along with Morgagni.

The compendia published in 1785, by C. T. Ludwig, and in 1796, by Conradi, and even the greater work of Voigtl (1804), so marked by literary industry and so serviceable withal, have not advanced science either by aptness of discrimination, by a judicious selection of matter, nor yet by any remarkable progress in the method of anatomical research.

Mathew Baillie's anatomy of morbid structures (translated into German by Soemmering, in 1794) is distinguished by greater depth of research into the fabric of organs, and both by its generalising tendency and its physiological character. These latter qualities are, however, still more decidedly impressed upon the aphorisms from pathological anatomy published at Vienna, by Velter, in 1805.

The most decided impulse was given to a right conception and application of pathological anatomy by Bichat in his general anatomy. Bichat founded upon the latter an especial physiology, or rather, blended the two. Pathologists, imitating this, endeavoured to reconstruct their science upon an anatomical basis.

France was the country in which this attempt was made in the most effectual manner; not that it was exactly the cradle of pathological anatomy, but that it was the land of all others in which men sought and found in it a solid foundation for medical knowledge. Such men were, amongst others, Bayle, Corvisart, Laennec, Dupuytren, Broussais, Cruveilhier, Rochoux, Lallemand, Riobé, Andral, Louis, Gendrin, Bouillaud, Billard, Rayer. It is true that one of these, namely, Broussais, disseminated an error from which his pupils cannot yet disentangle themselves, an error in which Brunonianism seemed once more to be trying its strength upon novel ground. On the other side, however, Laennec invented and carried out a method which ensures to him and to his work the acknowledgment and admiration of future ages.

In England many have, up to our own day, worked in a similar spirit. Amongst these, we may mention the names of Abernethy, Charles Bell, Astley Cooper, Hodgson, Farre, Wardrop, Howship, Baron, Hodgkin, Hope.

In Italy, on the contrary, and in Germany—if we except the impulse so decisively given in the same direction by the ingenious Reil—pathological anatomy has been upon the whole less cultivated, and has exercised less influence upon medicine. Accordingly, Germany and Italy have but few men to place in parallel with those of France; few to add to the names of Scarpa, Malacarne, Paletta—of J. F. Meckel, Otto, and (in industry and method, the essentially German) Lobstein.

It was reserved for Germany, at the present day, to establish a pathological anatomy and a method of working it out, partly independent, partly framed according to the best models of France. Under the auspices of German universality and analysis, this renovated science, emancipated alike from the systems of a by-gone age and from a vain eclecticism, has begun to incorporate itself with pathology in a way that promises both durability and brilliant progress, more especially in its natural alliance with German physiology, and under a consistent and rational standard of pathological chemistry.

Classification.—Just as there is a general and a special anatomy, physiology, pathology, so there must in like manner be a general and a special pathological anatomy. The former treats of general anomalies of organisation, the latter of the special anomalies of individual textures and organs.

All anomalies of organisation involving any anatomical change manifest themselves as deviations in the quantity or quality of organic creation, or else as a mechanical separation of continuity. They are reducible to *irregular* number [deficient or excessive formation], irregular size, form, position, connexion, colour, consistence, continuity, texture, and contents. They relate to the physical properties of the animal body and of its organs. The chemical properties, although not strictly pertaining to the field of anatomy, are too intimately connected with the physical to be suffered to remain in the background at the present day. The animal fluids bear a similar relation to anatomy. Their anomalies will be taken into account, so far as it may appear needful, under the appropriate heads. Those of the sanguineous fluid will, however, demand a separate chapter. This will come in at the conclusion of the general anatomy, in which a frequent reference to them will have previously demonstrated the indispensable nature of the enquiry, as a sort of connecting link between general and special anatomy. We shall thus have to discuss, in ten separate chapters, the anomalies of organisation. There are, however, a few general points which require some previous explanation.

I. The said anomalies, being simple alterations of the normal being and of its parts, appear as abnormal conditions, excluding the idea of an independent parasitic organism of disease.

II. No formation is incapable of becoming diseased in one or more ways. Several anomalies coexisting in an organ commonly stand to each other in the relation of cause and effect. Thus, deviations in texture very frequently determine deviations in size, in form,—and these again deviations in position. Deviations in position give rise to anomalies of volume and of texture.

III. Pathological anatomy, proximately concerned with anomalies of individual organs and systems—with local anomalies—has often reserved for it the task of revealing by experiment and deduction the existence of *general disease*, as also of establishing the mutual relations which exist between the two. The seat of general diseases may now be referred, almost without exception, to the blood [the fluids]. They appear, therefore, as anomalies of admixture or crasis, either primary or secondary.

IV. This demonstration of general disease is indeed a step in advance for pathological anatomy. It threatens, however, to mislead us into the error of exclusive, transcendental, all-pervading humoralism—into the error of denying all local disease, by deducing the latter in every instance from a corresponding general affection,—not but that many diseases really are but the localisation of a pre-existent general disease.

v. The existence of purely local—independent of general—disease, from the simplest inflammation—from blennorrhœa, to tubercle and cancer, we look upon as grounded—

(*a.*) In the self-vitality of organs, and their independent relations to the external world.

(*b.*) In the local influence of direct or reflected stimulation. Either directly, or through the medium of the nervous system, stimuli effect a local modification in the vital processes of absorption and secretion—in the interchange of matter,—an anomalous reciprocity between blood-vessels and their contents on the one side, and the parenchyma-engendering products, abnormal both in quantity and in kind, on the other.

Normal nutrition and secretion are no doubt mainly dependent upon a normal crasis; but they are also based upon the perfection of the specific vital action proper to individual parenchymata. Anomalous secretions often arise out of influences which modify the vital action of the parenchyma, and consequently its reciprocity with the unchanged gross material, the blood: as, for example, augmented or otherwise altered secretion of milk, produced by local irritation or by anomalous innervation, the effect of mental operations. In like manner, local diseases are but a consequence of qualitative and quantitative alienation of the textures and organs,—the formative material (the blood), notwithstanding its reciprocity with the latter, not becoming sensibly contaminated.

Influences, especially of a mechanical kind, are often so strictly local, that it would be far-fetched to derive all local disorder from a general causal disease. Even the latter would be but secondary,—a mere transfer of the alienation locally produced.

The existence of local diseases is further shown—

(*c.*) By direct evidence, where local disease is established, of the absence of any disease of the blood crasis.

(*d.*) In the curableness by topical remedies—extirpation, isolation, &c.—of local diseases, without their recurrence either on the same spot or elsewhere. The cure may even involve the simultaneous removal of a general disease consequent upon the local one, this having possibly acted as an anomalous instrument for the elimination of certain elements from the blood, exhausting it of certain essential constituents.

VI. Local disease extends beyond its original seat in various ways:

1. By contiguity. The affection spreads to the immediate vicinity of its original seat. This extension is favoured—

(*a*.) By uniformity of structure.

(*b*.) By intensity of disease.

(*c*.) By the nature of the malady. Certain diseases, such as tuberculosis and cancer, in their extension, spare no texture, whilst the typhous process upon the intestinal mucous membrane always finds an arresting formation in the sub-mucous areolar tissue.

2. The disease extends to remote formations, both similar and dissimilar. This mode of diffusion does not imply concurrent general disease, but proceeds, according to tolerably constant laws of sympathy, through the mediation of the nerves. It is greatly promoted, however, by a concurrent general disease, kindred in character with the local, causing it to increase, and multiplying the seats of the disease, as, for instance, in inflammation.

VII. The originally mere local is enhanced into a general disease of the same nature, or gives rise to one of different attributes. The former contingency may arise from the alienation of the peripheric nerves, productive of the local anomaly, being transmitted to the nervous instruments presiding over the circulation,—in other words, to the nervous centres, and in particular to the spinal chord and the ganglia. Or else the conversion assumes the substantive form of infection, the noxious matter evolved by the original local disease, or its products, being received into the circulation. This last event occurs where the products of the local disease exhaust the blood of certain ingredients, for instance, of fibrine, of albumen, of serum, of salts. To this class belong, in like manner, the anomalies occasioned by mechanical disproportion, such as the venous diathesis, cyanosis dependent upon disease of the heart or lungs, &c.

VIII. The presence of general disease may be the more safely inferred:

1, The more widely extended is the local disease over several uniform or dissimilar formations, and the greater its intensity;

2, The less the products of the local process are conformable with the character of the normal structures;

3, The less the extent and nature of the local disease, or of the structures involved, however important in the organism, suffice to account for the general appearances during life and after death;

4, The more anomalous, compared with the alienation of the solids, are the secretions and excretions; and,

5, The more the totality of the organism, in the absence of actual anatomical disturbance, seems cachectic and impaired.

6, The more marked is some anomaly in the circulating fluid, with respect to the quantity or quality of its component parts.

IX. General disease engenders in the most various organs and textures, according to their innate general or individual tendencies, either spontaneously or by dint of some overpowering outward impulse, a local affection which reflects the general disease in the peculiarity of its products. The general disease becomes localised, and, so to speak, represented, in the topical affection.

X. A general disease not unfrequently finds in its localisation a perpetual focus of derivation, with seeming integrity of the organism in other respects. Recovery may, after a lengthened process, eventually take place through the exhaustion of materials at the local vent. Forced extirpation, on the contrary, or insulation of the locality, generally aggravates to a high degree the general disease, multiplying its points of localisation.

XI. The disease has, even anatomically speaking, its stages of incipiency, increment, acme, and decline.

XII. The terminations of disease are, in like manner, subjects for anatomical research.

1. The issue of local disease in health consists either in the perfect re-establishment of the normal condition, or else in

partial recovery; more or fewer important residua and sequelæ of the disease, not incompatible with a tolerably fair state of health, remaining entailed. Thus the previously diseased organ may have lost substance, or more or less its natural texture; or it may have suffered changes in form or in position, or interruptions of continuity.

2. The issue of one general disease in another general disease [metaschematismus] is frequent. Anatomical research proves, and chemical analysis will still more clearly demonstrate, that it is far more frequent and varied than would appear from mere clinical observation. This is taught in an especial manner in the mutual exclusion of different morbid processes, which seem to succeed each other, when in full vigour, sometimes almost by a necessary sequence. Thus dropsy may succeed to the exhaustion of fibrine and the excretion of albumen, cancer to tubercle, &c.

3. Transition by so-called metastasis often becomes the subject of the scalpel. It comprises various conditions:

(*a.*) The localisation of a general disease at an unusual spot. It has the character of a vicarious or supplementary crisis. Instances are afforded in skin eruptions, and especially in the secondary typhous processes.

(*b.*) Topical processes constituting the localisation of a metaschematism, with which, as in the former instance, a general disease concurs. Such metastases occur more particularly in the sequel of typhus, and in the shape of inflammation, suppuration, gangrene, in both external and internal organs. They represent the localisation of a general disease consecutive to the original typhous process.

(*c.*) Local processes, with the development of which the general disease is essentially abated, or thoroughly exhausted and extinguished. They are frequent, and deserve alone to be designated as *metastases,*—metastases in a restricted sense. They are either just sufficient vents for the general disease, and are only cured when the latter is subdued; or they heal spontaneously, the dyscrasis having, through their agency, become exhausted.

(*d.*) When, owing to whatever cause, a local disease has been checked in its development, it subsides only to reappear in another part, often with augmented force, and with the supervention of a new general disease, or the aggravation of one already existent.

4. *Issue in death.* Diseases are mortal for the most part,

(*a.*) Through exhaustion of power and of organic matter, tabescence, loss of fluids.

(*b.*) Through the suspended function of organs essential to life; through palsy; through sudden and extensive displacements; through hypertrophy, atrophy, diseases of texture.

(*c.*) Through vitiation of the blood and palsy of the nervous centres arising out of the conflict between contaminated blood and nervous medulla.

XIII. Where several diseases coexist in an individual, they are in part *primary*, in part *secondary* and subordinate to, although homologous with, the former. Again, they are partly sequelæ and residua of antecedent disease, as in the case of atrophy of the brain consequent upon apoplexy, upon encephalitis.

XIV. Very dissimilar anomalies may coexist in one individual, as mere local affections or complications. *Combination* or *exclusion* result only in the case of heterogeneous diseases founded in or determining a dyscrasis: for example, cancer and tubercle, organic heart disease and tuberculosis. The study of these two relations opens a rich field of promise for the furtherance of accuracy in diagnosis.

XV. The import of a disease bears a direct ratio to the worth of the organs attacked: take, for example, hypertrophy in different muscular organs.

XVI. With reference to the period during which anomalies originate, we have to distinguish *congenital*, or such as have become established during intra-uterine life, and *acquired*, or such as have arisen during extra-uterine life. The former comprehend *primitive anomalies.*

XVII. Primitive anomalies comprise *malformations.* These are deviations of the organism, or of an organ, so intimately blended with its primary development, as to occur only at the earliest periods of embryonic life, or at any rate before that of mature fœtal existence.

Malformations, when inconsiderable and harmless to the individual, are termed *lusus naturæ, variation, defect of formation, malformation;* when more marked, *deformity;* when excessive, *misbirth, monstrosity, monster.*

Despite some progress made in this field of late years, the genesis of malformation is still veiled in much obscurity. The opinions of modern physiologists on this point may be collected under two heads. According to the one section, the malformations are referable to a primitive malformation of the germ. According to the other, to various influences affecting the germ in the progress of development.

The former opinion resolves itself into that of the *ovists* and that of the *spermatists.* Those believed in the foreshadowing of the malformation in the ovum; these regarded it as dependent upon the spermatozoa as embryones.

At this day both theories are rejected as inapplicable to a vast number of malformations which unquestionably do originate during the development of the germ. Still the malformation might be founded in the nature of the ovum and of the sperma, although neither of these constitutes the embryo. The frequent recurrence of the same malformations out of the same parents, and the hereditary character of these anomalies, render this not improbable. We might further advert to the nature of certain malformations—inversions, duplicate formations, for instance—in which the fusion of two germs, and the bisection of a single germ, during their development, are neither of them quite conceivable.

The second of the aforesaid propositions embraces several hypotheses.

(*a.*) The oldest and most popular attributes the malformation to a sudden and forcible impression wrought upon the mother (Verschen). The question whether mental emotions do influence the development of the embryo must be answered in the affirmative. Instances undoubtedly have occurred of such maternal impressions—fright more particularly—when violent, giving rise to malformations. Seeing that many malformations originate in an arrest of development, and how frequently the former bear a certain resemblance to various animals, it is just conceivable that the development of the embryo may be so arrested by maternal emotions as *accidentally* to occasion a likeness between the object that produced the impression and the resulting malformation.

(*b.*) A second doctrine derives malformation from external mechanical influences, such as a blow, a thrust, a fall, &c., suffered by the mother; mechanical obstacles to the passage of the ovum through the Fallopian tubes, and to its growth in the womb; excess or deficiency of liquor amnii; restriction of space for the fœtus; the formation of false membranes within the cavity of the amnion, &c. Although F. Meckel dissents from this doctrine, we are not quite prepared to relinquish it ourselves.

(*c.*) A third opinion assigns, as the cause of malformations, disease of the fœtus.

Disease arrests the development of fœtal formation with respect to growth, shape, position, and texture of particular organs, indirectly embarrassing the expansion of neighbouring organs, or it may be, causing their destruction. A common disease having this tendency is dropsy, as preventive of union or closure, and productive of disjunction or fissure. Inflammation and its consequences may be mentioned under the same head.

The conditions of encephalocele, of hemicephalus, of anencephalus, and of spina bifida, being obviously due to dropsy, are beyond the pale of arrested formation. Certain anomalies of the peritonæum and of its viscera, formerly reckoned as genuine instances of arrested formation, have been shown by Simpson in certain instances probably to result from fœtal inflammation. Some malformations of the heart, especially defects in its septa, we hold to be owing to fœtal endocarditis, and to consequent coarctation of the heart's orifices.

The number of malformations, however, to which this doctrine fully applies, is as yet very small. Amongst those which do not admit of such an explanation are *duplicate formations*, and the great majority of malformations designated by the term *malposition*.

(*d.*) The fourth proposition—which the conntenance given to it by Wolff, by Tiedemann, and especially by J. F. Meckel, has caused to be the more generally received one—has formed the groundwork for an elaborate scientific inquiry into the subject, in especial connexion with the history of development. This sets forth that *most malformations represent certain stages of the development of the embryo and of its organs, at which stages formation has stopped short, or from which ulterior development has ceased to follow the normal type.* The *malformation* is therefore essentially an *arrest of development.*

This theory of malformations is in a great measure correct. Still it does not attempt to explain the *cause* of the arrest, which may be one of those already enumerated, be it concerned with the germ, with sickening of the embryo, with mechanical influence, or with mental emotion.

A good classification of malformations is, owing to the difficulty of establishing a principle of division generally applicable, as yet wanting. If we attempt to classify them according to external form, we meet with a barrier in their multiplicity. Another obstacle consists in this, that where several malformations coexist in the same individual, they must needs all be classified according to the one most pronounced, and the designation be therefore partially incorrect. A classification founded upon the occasional causes is impracticable, since the same malformation may originate from various causes. If we take for the basis external form and cause conjointly, the classification becomes bereft of logical unity.

It is desirable that we should become acquainted with the principles upon which the more remarkable classifications hitherto propounded are built.

Passing over the older classifications, we should except that of Buffon, as it forms the groundwork upon which almost all the later ones are modelled. Buffon divided malformations into three classes: 1, malformation with excess; 2, with deficiency; 3, with inversion or perverse site. To this classification we may subjoin that of Blumenbach, under the following four heads: 1, fabrica aliena; 2, situs mutatus; 3, monstra per excessum; 4, monstra per defectum. These were followed by Meckel in his division of malformations, as follows: 1, malformation from deficient plastic power; 2, from excess of plastic power; 3, from deviation of the organs in respect to their natural form; 4, malformations characterised by ambiguity of sex—hermaphroditism. This distinction of hermaphroditism from other malformations constitutes the great defect of Meckel's classification.

Breschet has, in his classification, broken up Buffon's first class into two, by separating duplicate formations from malformations per excessum. The four orders of his classification are: 1, ageneses, devious formations with diminution of plastic power; 2, hypergeneses, with augmentation of plastic power; 3, diplogeneses, devious formations with the fusion of germs—duplicate formations; 4, heterogeneses, with alien character of the product of generation. The further division is as follows:

The first order, ageneses, breaks up into four species.

(*a.*) Agenesie, absence—defective development. It is either partial, as in hemicephalie, aprosopie, acephalie, apleurie; or it is general, as in microsomatie (dwarfishness, cretinism).

(*b.*) Diastematie, cleft formation at the median line. It is subdivided according as it affects the head or the trunk, into diastemencephalie, &c., and diastematosternie, &c.

(*c.*) Atresie.

(*d.*) Symphysie, coalition, fusion.

The second order, *hypergenese,* presents two species, according as individual parts or the entire body are concerned. To the former species belong macrocephalie, macroprosopie, &c.; the latter consists of macrosomatie (giant growth).

The third order, *diplogenese,* is divisible into *external,* through fusion or adhesion, as in diplocephalie, diplothoracie; and *internal,* through penetration (per penetrationem).

The fourth order has three species.

(*a.*) Deviation as to site, either of the entire organism [extra-uterine pregnancy] or of individual organs [ectopie].

(*b.*) Deviation as to number, polypædie—the coexistence in the uterus of several fœtuses.

(*c.*) Deviation as to colour—leucopathie, cyanopathie, cirrhopathie.

In this arrangement, the distinction of diplogenesis from hypergenesis is based upon the unproved doctrine of the fusion of two germs constituting duplicate formation. To the order, heterogenesis, are referred deviations which ought not to be designated as malformations.

One of the best known classifications of late years is that of the two Geoffroy St. Hilaires, father and son, who handle malformations, according to the natural method, under the term teratology (from τερας, monstrum).

Malformations are simple and *complex—anomalies simples et complexes.*

The simple—hémitéries—are either so-called variations, lusus naturæ, where the anomaly is slight, causing neither disturbance of function nor deformity; or else defects of conformation, malformations in a restricted sense, where, however trifling the anatomical deviation, they impede or preclude the exercise of one or more functions, or occasion deformity.

They are divided into five classes, the anomaly being respectively concerned with—

1, *Volume*, as regards size, both of the body generally, and of its individual parts;
2, Form;
3, Structure and coloration;
4, Disposition;
5, Number and existence, that is absence or presence of parts.

These classes, according to extent and to degree—according to the kind of malformation, are divided into *orders*, and these again distinguished according to the regions, systems, and organs involved. Thus the first class comprises the four orders—general dwarfishness and general gigantism, partial gigantism and partial dwarfishness of proportions. The fourth class includes the five orders—displacement, preternatural union, preternatural connexion, sept-formation, disjunction, &c.

Complicated anomalies are classed in three subdivisions.

1. Heterotaxies (ἑτερος and ταξις). Anomalies important in an anatomical sense, but neither visible externally nor obstructive of any function. In mankind they comprise but

one order, namely, *lateral* inversion of viscera (inversion splanchnique).

2. Hermaphroditism.

3. Monstrosities. Anomalies very considerable in degree, and consisting in a faulty anatomical arrangement greatly deviating from the type of the species, externally visible, and obstructive of one or more functions.

These last are divided into three classes,—into simple, double, and triple. The next division into *orders* is arranged according to physiological characters; the subdivisions then following, according to *tribes, families,* and *species*. Thus, simple *monstrosities* resolve themselves into three *orders:*

1. *Autosites,* in which independent, progressive development is possible. They are capable of thriving for a shorter or longer period extra uterum.
2. *Omphalosites,* in which mere passive nutrition is effected through the placental circulation. They are altogether very imperfect, more especially in relation to symmetry of the two sides of the body.
3. *Parasites;* shapeless masses, deficient even in an umbilical cord, adherent to the sexual organs of the mother, and nourished at their cost.

The first order, *autosites,* is divided into four tribes: the first tribe into two families; *ectromeliens,* malformations with deficiency of the extremities, with the varieties—phocomèle, hemimèle, ectromèle; and *symeliens,* fusion of members, with the varieties—symèle, uromèle, sirenomèle. The second tribe has the single family *celosomiens,* prolapsus of viscera and imperfect anterior closure, anterior fissure, eventeration, with the varieties — aspalasome, agenosome, cyllosome, schistosome, pleurosome, celosome. The third tribe embraces the three families: *exencephaliens,* imperfect brain, extra cranium; *pseudoencephaliens,* slender rudiments of brain, with deficiency of a large proportion of the skull; and *anencephaliens,* complete absence of the brain and skull, with their varieties. The fourth tribe resolves itself into two families: *cyclocephaliens,* arrested formation and fusion of nose, eyes, and upper jaw; and *otocephaliens,* approximation and blending of the ears, with arrested development of the base of the skull and brain, and concurrent malformation of the apparatus of mastication—with their varieties.

The second order, *omphalosites,* has two tribes, with three families and their varieties. The first tribe comprises the two families, *paracephaliens,* rudimental head formation, asymmetria and absence of extremities and of many vegetative organs; and *acephaliens,* complete absence of head, with its varieties. The second tribe has the single family of *anidiens,* reduction of the entire organism to a membranaceous sac inclosing various soft formations and sundry blood-vessel ramifications.

The third order, *parasites,* has the one family *zoomyliens,* rudimental embryo in the abdomen, in the genitals, &c., with a kind of zoomyle.

Double monstrosities are of two orders.

1. Double autositic monstrosities—fusion of two autosites.
2. Double parasitic monstrosities—union of an autosite with an omphalosite or parasite.

The first order (double autositic) embraces three tribes.

The first tribe subdivides into two families: *eusomphaliens,* the union of two nearly perfect organisms, each possessed of a normal umbilicus and umbilical cord, with several varieties; and *monomphaliens,* the union of two organisms having one umbilicus in common, also with several varieties.

The second tribe includes the two families, *sycéphaliens,* fusion of head and trunk; and *monocéphaliens,* two trunks with one head; with their varieties.

The third tribe contains the two families, *sysomiens,* single trunk with double head; and *monosomiens,* mere vestiges of duplicity about the head; with their varieties.

The second order (double parasitic) comprehends three tribes.

The first tribe has two families: *heterotypiens,* parasite and autosite united about the umbilical region, with varieties; and *heteraliens,* with the single species, epiconu, parasitic head upon the vertex (capitis) of the autosite.

The second tribe blends the two families of *polygnathiens,* imperfect head implanted in the maxillary apparatus of the individual; and polymeliens (μελος, membrum), the parasite consisting solely of extremities and adjuncts; with varieties.

The third tribe has but one family, the *endocymiens*, a parasite inclosed within the autosite.

The *triple monstrosities* admit of the same distinction as the *double*, namely, into *triple autositic* and *triple parasitic.*

The doctrine of the two Geoffroys, respecting malformation, frequently errs in their having neglected to adopt for its basis the natural laws of development. The system is even not devoid of logical inaccuracies, nor sufficiently compendious for practical use.

Another well-known German classification is that of Gurlt. He divides malformations, generally, into the three classes of malformation in one body, or simple monstrosities (monstra simplicia or unicorporea); double or twin monstrosities (m. duplicia or bigemina); and threefold or trigeminal monstrosities (m. triplicia or trigemina).

The first class is divided into six, and, including hermaphrodite forms, into seven orders.

1. Malformation from deficiency of parts.
2. From minuteness of parts.
3. From preternatural fissure.
4. From non-perforation and from fusion of parts (atresia and symphysis).
5. From preternatural form and site.
6. From extraordinary number of parts.
7. Hermaphrodites.

The second class has two sub-divisions.

- I. Double malformations from coalition.
- II. Double malformations from implantation.

The first sub-division breaks up into four orders.

1. Coalition without separation at either end of the body.
2. Coalition with separation at the upper end.
3. Coalition with separation at the lower end.
4. Coalition with separation at both ends.

In this classification, as in those of Breschet, double formations are made distinct from malformations through excess of parts.

The most recent classification is that of Otto. It approximates to those of Buffon, Blumenbach, and Meckel. It arrays malformations in three classes.

First class.—Monstra deficientia, furnishing three orders.

1. M. perocephala, deficient in some one portion of the head, of which there are seven species.
2. M. perocorma, malformations with deficient vertebral column.
3. M. peromela, deficient development of the extremities.

Second class.—Monstra abundantia. These are divided into two orders.

1. M. ex duobus coalita.
2. M. luxuriantia.

Third class.—Monstra sensu strictiori deformia. It resolves itself into four orders.

1. M. fissione deformia.
2. M. coalitu singularum partium deformia.
3. M. atresiâ deformia.
4. M. morbis manifeste deformia.

To this system there is much to object: for example, that perverse site, that anomalies with respect to blood-vessels, and certain hermaphrodite formations have no place in it; that fissures and atresiæ are not admitted as monstra deficientia; that monstra abundantia are ascribed to coalition, &c.

Lastly, Bischoff begins by showing that for a classification of malformations the anatomical character alone can be made available, and that the physiological principle is here altogether inapplicable. Having then pointed out the proper method of determining the anatomical character—the diagnosis—of a malformation, he proceeds to build up the following system, which, generally approximating to those of Buffon and Blumenbach, frequently differs from both in detail, whilst, by its rigid adherence to anatomical principles, it seems to take the only admissible ground.

First class.—Malformations deficient in some essential attribute of their kind.

Second class.—Malformations possessing more than pertains to the standard of their kind.

Third class.—Malformations, the organisation of which does not conform with the standard of their kind, but without either the deficiency or the superfluity just referred to.

First class.—The causes to which the malformations, here under consideration, are due, may be very various. In many instances we are justified in regarding them as products of imperfect conception, whether the fault lie in imperfect formation of the ovum or in anomalous quality of the semen. At this day, however, so much in this assumption is still hypothetical that we are compelled to deal with it cautiously, addressing ourselves, where it is possible, to other causes, more especially to interrupted evolution of an organ out of its germ, or to its development being impeded through external influences, such as impressions wrought upon the mother; destruction of the organ, in the progress of its development, through disease, particularly through dropsical accumulation; finally, destruction of an organ through mechanical influence—for example, the amputation of a limb by means of the umbilical cord or a pseudomembranous formation within the ovum, &c.

This class comprises the following orders:

1. Deficiencies in a stricter sense.
2. Malformation from diminutiveness of parts.
3. Malformation from coalition (symphysis).
4. Atresiæ.
5. Cleft-formation.

Second class.—Here we meet with a regular progression, from the supernumerary bone or finger up to the development of two perfect individuals, united only at one part. The series of this progression is so graduated and so complete, that Bischoff regarded it as impracticable, even on anatomical grounds, to make any break in the respective formations, although Breschet and Gurlt have done this by distinguishing formations possessing single supernumerary parts with a single head and trunk, from those in which these latter are twofold, and which they denominate twin-malformations. This distinction is, however, based upon a physiological principle in itself objectionable. It is only to the former species of malformation that we assign excess of plastic vigour as the source of the supernumerary parts; whilst true twin-formations are referred to the fusion or coalition of double primitive germs, implying deficiency of plastic vigour, inasmuch as each germ individually must be imperfectly developed.

This class comprises the following orders:

1. Malformations from superfluity of single parts, with a single head and trunk.
2. Twin malformations with double head and trunk.
3. Double malformations from implantation.
4. Triple malformations.

Third class.—Its defect is that its characteristics are principally of a negative kind.

The objects comprised in this class being very numerous, their probable sources are in a corresponding degree various, For several formations no other cause can be assigned than an anomaly of plastic activity originating in some primitive configuration of the germ; in a few other instances disease may be assumed as the cause; the majority, however, will be explicable on the ground of arrest of development.

This class contains the following orders:

1. Change in the position of organs.
2. Deviations in the form of organs.
3. Deviations in the origin and the disposition of the arteries and veins.
4. Hermaphrodites.

As, according to our plan, all malformations will be considered under heads corresponding with the classes and orders of the above system, and as we shall also follow Bischoff in our physiological notice of the species, an occasional reference to this system will, for the present, obviate the necessity for a further enumeration of the species belonging to each order.

On the other hand, we have here to notice, in a general way, the laws which nature observes in the production of malformations, so far as a general working out of this subject has revealed them to us.

1. The worst malformation is never so anomalous as not to bear the general characters of animal life, and the external semblance of the particular class of animals to which it belongs.

Even an individual organ never departs from its normal character so completely that, amid even the greatest disfigurement, this character should not be cognisable.

2. Deviations from the normal are, then, confined within certain limits, and this applies in an especial manner to anomalies of position. Although that which should lie on the right may appear on the left, and the converse—the abdominal organs occupy the thorax, and the thoracic the abdomen—the brain has never yet been found in the chest or abdomen, nor the kidneys within the skull. The natural history of development reveals the cause,—different organs and systems being developed out of different layers of the germ; those pertaining to the same layer may indeed err as to their locality, but in no instance will an organ pertaining to the animal, become evolved out of the vegetative layer of the germ, nor the converse. Fleischmann calls this the law of localities (lex topicorum).

3. To this we may add, that certain conjunctions between organs, for example, the aorta and the intestinal canal forming a single tube in common, never occur; but that, as a rule, homogeneous or kindred parts alone unite, a law termed by Fleischmann the law of individuality (lex proprietatis).

4. The excessive development of one part determines the imperfect, retarded development of another, and the converse. Meckel having laid it down as the next thing to a law, that a preponderance of one organ is associated with the retarded growth of another, Geoffroy St. Hilaire has invested this law—as the law of compensation (loi de balancement)—with the most ample significance and extension. The said law has in reality sundry facts for its foundation; it is alleged, in particular, that individuals having on one hand or one foot a supernumerary finger or toe, are often found wanting in a finger or toe on the other foot or hand. A fœtus described by Neumann had on the left foot only the great toe, but, on the right, eight toes, the eighth being cleft. Segala's fœtus had no thumb to the left hand—to the right, two; it had on one side eleven ribs only, but thirteen on the other. In cases where more or fewer important parts are wanting or imperfectly developed, we often find supernumerary fingers and toes; for example, in anencephalia, cyclopia, spina bifida, hare-lip, cleft abdominal pa-

rietes, &c. In the siren-malformation there is, according to Meckel, always an excessive number of vertebræ and of ribs. In acephali, deficient in heart and liver, the kidneys are asserted by Elben to be preternaturally developed. On the other hand, in the double formation of individual parts, others are frequently imperfect: thus, bitruncate malformations are frequently acephali, whilst the bicephalous have often spina bifida; and in either case sundry other organs besides have suffered an arrest of development, being deficient in abdominal parietes, the intestinal canal being imperfect, the urethra imperforate, or cloacal malformation present. Meckel has even made this law apply to different children of the same parents: one girl had on each hand a supernumerary finger, her sister had *two* fingers wanting to one hand.

Meckel, rightly, we think, rejects the assumption of a *law* of compensation, where compensation is so far from general, and admits only that malformations are often influenced by a law common to organised bodies.

5. Not every organ or part is in an equal measure obnoxious to malformation. According to Meckel it is far more rare in organs supplied by cerebro-spinal nerves (muscles, larynx, lungs), than in those supplied by the sympathetic (the digestive, urinary, generative). The vascular system is, however, most liable of all.

6. Certain malformations affect certain organs. Thus, it is an admitted fact that formations resulting from the vegetative and the vascular layer of the germ seldom multiply, compared with those which result from the animal layer. Instances of multiplied heart, lungs, intestinal canal, uropoietic and generative organs, are far more rare than of multiplied head, organs of sense, extremities, &c.

7. Whilst certain malformations are about equally frequent in both halves of the body, certain others affect by preference the one or the other side of the upper or the nether half of the body. Where the vertebral artery originates immediately from the aorta, "this," observes Meckel, "happens invariably on the left side." Cleft lip and cleft palate are commonly found on the right side. Malformations from superfluity are much more frequent in the upper than in the nether half of the body. Thus, bicephalous monsters with a single trunk are more fre-

quent than monocephalous with double trunk; supernumerary fingers than supernumerary toes. In like manner, anomalous blood-vessels are more common in the superior extremities than in the inferior.

8. Female malformations are, by all accounts, much more frequent than male. A reason for this cannot at present be assigned.

Of the hereditary nature of malformations, and their repetition in children of the same parents, Meckel has collected numerous examples. The entail is transmitted equally through the male and through the female line. Meckel adduces an instance of a man with six fingers to each hand and six toes to each foot transmitting the same malformation to his eldest son, whose three sons again were born with precisely the same redundant organisation.

Various and manifold as are the forms of monstrosity, some of them recur with such uniformity of type, as to constitute a regular series. This applies to every organ, each being especially liable to some particular kind of malformation. This circumstance is of great importance in summing up the causes of malformations. It indicates that, in the majority, not an extrinsic, accidental cause prevails, but an intrinsic one, inherent in the laws of germination and development.

With the aforesaid laws, derived more immediately from malformations, a mistaken attempt has been made to couple two other special laws:

1. The first being that of Serres, according to whom the development of an organ altogether depends upon the development of the blood-vessels, and especially of the arteries. Conformably herewith, imperfect development, or the absence, or again the excess of an organ or part, would be a consequence of the insufficiency, or the absence, or again of the preternatural development of the supplying artery. But even were the fact altogether true, the cause of the defective or excessive development of the artery would still remain to be accounted for. The relation, therefore, not of dependence, but merely of correspondence between the degree of development of the malformed organ and of its supplying vessels, would be proved, as the rule, and even this subject to occasional exceptions. Bischoff, however, regards as decisive the direct observation that, in their

rudiments, organs are immediately evolved out of the germ, previously to their being furnished with blood-vessels; the constituting cells *subsequently* becoming metamorphosed in such wise, that, out of one portion blood-vessels and blood, out of another the other (secondary) elements of the organ are derived.

2. According to the second law, the nerves are substituted for the blood-vessels, as the media of development. Tiedemann has shown that, with the absence of certain nerves is coupled the absence of their dependent organs; that in all monstrosities with excess, a corresponding relation is demonstrable in the nervous system; and, again, that in malformations with coalition of organs, the fashion of this union is exactly imitated by the supporting nerves. On the other hand, the natural history of development has shown that the central parts of the nervous system constitute the first vestiges of the embryo, being thrown out cognisably as such by the germ. Upon such grounds, the opinion has been formed that, like the normal, so also the anomalous development of the different organs of the embryo is dependent upon the normal or anomalous development of the nervous system. Against this view the objection hinted at in the last paragraph might again apply.

XVIII. The disposition to different diseases varies according to age, sex, climate, &c.

Thus, aneurism belongs chiefly to manhood and advanced age, rickets exclusively to childhood; the fœtus labours under anomalies proper to primary development alone—namely, malformations. In childhood tuberculosis attacks, preferably to all other parts, the lymphatic glands, the brain; at and beyond the age of puberty, the lungs. The female sex greatly favours the occurrence, in the sexual system, of cystoids, of cystosarcoma, of the majority of cancerous growths. Under certain climatic relations, tuberculosis is rare,—intermittent fever, hypertrophy of the spleen, frequent; under the tropics the ossification of arteries is said to be extremely rare. Again, particular regions and parts of the body manifest different dispositions with respect to the frequency of congenital or acquired anomalies. Thus, Portal pronounces apoplexy to be more frequent in the right corpus striatum than in the left,—

pneumonia is more common in the right lung than in the left. The arteries of the inferior extremities are infinitely more obnoxious than those of the superior to ossification and to spontaneous aneurism; the veins of the lower half of the body are almost exclusively subject to varix. Malformation from excess appears more frequent in the upper half of the body, malformation from coalition more frequent in the lower half; variations in the course of blood-vessels are more rare in the inferior extremities than in the superior.

PATHOLOGICAL ANATOMY.

CHAPTER I.

ANOMALIES IN RESPECT OF THE NUMBER OF PARTS.

These consist in *diminution* or *augmentation* of the normal number of organic parts. It is not rare for both to be found united in one individual, one part presenting a deficiency, another, in virtue of the law of compensation, an excess of formation. Thus, monstrosities, in which otherwise deficiency predominates, will exhibit a superfluous finger or toe; double twin malformations, on the contrary, deficiency in various parts.

Deficiency or absence of individual parts, or diminished number of plural organs, are frequent,—for example, the absence of entire extremities, of individual fingers and toes; amongst the viscera, of one of the kidneys. It is either *congenital* or *acquired*. In the former case it includes malformations with *deficiency, in a stricter sense*. There is scarcely any part that has not been found wanting, without detriment to the entirety of the rest of the body. In this respect, however, certain relations of sympathy may not be overlooked, by virtue of which the absence of one part is paired with that of another part. Thus, in acephali the heart is almost always absent, very commonly, too, the entire thoracic viscera, together with the liver, the spleen, the pancreas,—an example of compliance with rule perfectly inexplicable; for neither can development from an identical germ, nor functional dependence of the organs here be argued. Occasionally so many parts are absent at once, that nothing is born save a head, a single extremity, or a shapeless mass. The absence of parts is very often, though by no means invariably, due to arrest of development.

The principal kinds of malformation that pertain hither are the following (Geoffroy St. Hilaire, Gurlt, Bischoff):

1. *Amorphus, anideus.* A shapeless mass consisting of cutis, areolar tissue, fat, and a few bones; is never found but in association with a perfect twin. It probably results from an early destruction of the germ.

2. *Acephalus.* The head alone may be wanting, or with it more or less of the trunk, so that, in fact, nothing may be present save a pelvis with the inferior extremities, or with one of these only. It is for the most part associated with a twin. Even though the trunk be present, the heart is usually absent; the respiratory organs probably always; liver, spleen, and pancreas commonly; stomach and intestinal canal are generally very defective; the uropoietic and generative organs are mostly present, though incomplete. It is in some instances perhaps deducible from injury to or destruction of the germ, or from the disturbance occasioned by a twin.

3. *Pseudacephalus, paracephalus.* Rudiment of head, with the rest of the body either entire or defective. These are sometimes twin cases, and are for the most part the consequence of hydrocephalus.

4. *Aprosopus.* The face, and especially the eyes, nose, and mouth, wanting. The skull is diminutive, and exhibits the ears coalescing, either in front or above. The brain is always very defective. The pharynx terminates, superiorly, in a cæcal sac. Here again, probably, destruction—splitting of the medullary tube, as also of the dorsal plates, at their anterior part, takes place at an early period. Hence the non-development of the anterior brain-cell with eyes and nose, and in like manner the superior arches of the cranial vertebræ, of the parietal and of the frontal vertebræ, and the consequent inclining towards each other of the temporal bones for the closure of the vertebræ. Hence, probably, also, the non-development of the anterior visceral arches, involving absence of the inferior maxilla and of the facial bones, with anterior junction of the external ears, which emanate from the second and third visceral arches.

5. *Microcephalus.* Diminutive, incomplete head; similar to the foregoing, except that the inferior jaw is present, proving the development of the first visceral arch.

6. *Anophthalmus.* Absence of both eyes or of one eye

only. Eyelids and lachrymal organs are present, although often defective—coalescent. Perhaps for the most part dependent upon dropsical destruction of the eye-vesicles, rudiments of the optic nerves being commonly discoverable.

7. *Absence of the eyelids;* an arrest of formation, these organs being of later development.

8. *Absence of iris;* in like manner, an arrest of formation.

9. *Anotus.* Absence of the external ears,—deficient development of the external portion of the first visceral groove.

10. *Brachyrhynchus.* Shortness of nasal prominence, owing to deficiency of intermedial jawbones.

11. *Acormus.* One rudimental head with a regular twin, or with a triple birth. Most probably due to mechanical hindrance to development, and to destruction occasioned by the other fœtus or fœtuses.

12. *Oligospondylus.* Absence of vertebræ or of semi-vertebræ, is owing either to an anomaly of original germinal development or else to the coalescing of two or more vertebræ or semi-vertebræ.

13. *Anaedæus.* Absence of the entire generative apparatus, or of the external organs of generation only; very rare as an independent malformation, where the individual is otherwise normally formed. It is an arrest of development, the said parts not being evolved out of the germ.

14. *Peromelus* and *micromelus.* The limbs are wanting or maimed. It is frequently an arrest of development; it may, however, result from mechanical influence,—from the severing of members through strangulation.

15. *Phocomelus.* Monstrosity with phocal extremities, the hands issuing directly from the shoulders, the feet from the pelvis, whilst the intervening parts are either wanting or merely rudimental. It is an arrest of development often dependent upon hydrocephalus or spina bifida.

16. *Parosomus.* Various deformities caused by the absence of individual parts.

17. Absence of various individual organs of the thorax or abdomen, of a lung, the liver, the spleen, the stomach or its blind sac, a portion of intestine, &c. It is either an arrest of development or the result of morbid destruction.

A particular kind of diminution of number consists in the symphysis or fusion of kindred formations; fusion of two fingers, toes, ribs, vertebræ, of the inferior extremities, of the kidneys, obliteration of the uterus. A deficiency of various grades is included under *cleft-formations.*

The acquired absence of particular parts is the result of mechanical influence or of disorganisation. To the former kind belongs maiming by accident or design; for example, amputation, extirpation, and the like, which sometimes greatly resemble certain congenital defects. To the latter kind is to be referred the wasting of various organs; for instance, from spontaneous, primitive atrophy, from that consequent upon inflammation, from destructive suppuration or gangrene.

Preternaturally augmented number of parts is very frequent, and commonly congenital. It occurs in every variety of grade, from the duplication of individual diminutive parts to that of the body almost in its totality. These various degrees constitute a series, the gradations of which are so regular, that it would appear forced to divide malformations of this class into such as with a single head and trunk present duplicates of individual parts, and into such as at the same time possess double or triple head and trunk. Nor does such a distinction derive much support from the assumption that the former are due to an excess of plastic activity, the latter to the primordial existence and the fusion of twofold germs.

Bischoff (with Winslow, Haller, Meckel) opposes the following weighty arguments to this distinction, as also to the assumption of twofold germs and their fusion (Lemery, Breschet, Gurlt, Chaussier, and Adelon).

1. The malformations of this class, from the supernumerary finger or toe up to the development of two perfect embryos united only at one point, constitute so complete and uninterrupted a series, that it would be in the highest degree forced to assign for the one and for the other opposite causes, namely, excess of formative activity, and again fusion with deficiency of plastic power. Still no one can hold a supernumerary finger or toe to be due to the fusion of two embryos.

2. In twin malformations, none but the same organs, systems, or parts, whether internal or external, ever coalesce. It is invariably thorax with thorax, abdomen with abdomen, head with head, breech with breech. Again, brain is ever found

coherent with brain, blood-vessels with blood-vessels, intestine with intestine, &c.; never trachea with œsophagus, nerves with blood-vessels, and the like. This is a fact, the constancy of which precludes its explanation on the ground of accidental fusion from external causes.

3. Twin malformations manifest, for the most part, a change not limited to the parts immediately united, but pervading the entire organism. Such a change could hardly be brought about by accidental fusion.

4. Twin malformations always recur with great uniformity of character. Are external causes likely always to combine after the same fashion?

5. Again, twin malformations often recur out of the same mother, and the tendency to them is hereditary. There is neither proof nor probability of an abiding external cause,——for instance, in the maternal organs of generation.

6. At no period of development is a mechanical fusion of the ova and embryos probable; indeed, our present knowledge respecting the nature and earliest development of the ovum renders such fusion highly improbable. The pellucid zone or yelk-bag is in the highest degree unfitted for it; nor is it at all possible to press two ovula against each other in such wise as to occasion the fusion of their yelks, of their germinal vesicles, or of the zonæ pellucidæ of the latter. In the Fallopian tubes and the uterus, however contracted, the ovula undergo no such risk, even in multiparous animals, where they lie densely grouped together. After the embryos have become developed and shrouded within their amnia, their coalition ceases to be even conceivable. These membranes would have to undergo previous rupture; for the occurrence of twins within a single amnion is too rare and too inexplicable to be here taken into account. How slight is the disposition amongst various embryos to coalesce is shown by those cases of twins, in which, owing to confined space, the one is almost flattened by compression, without any coalition having taken place.

It results from the above that the aforesaid division of malformations with supernumerary parts, and the assumption of twofold germs and their coalition, are inadmissible. The task, therefore, still devolves upon us of explaining this class of malformations. Accordingly we have to observe that:

1. The cause might consist in an anomalous formation of the ovum in its unimpregnated state. Bischoff adduces in favour of this view the occurrence of double yelks, as also the aforesaid hereditary character of twin formations and their recurrence out of the same mother. This cause would, however, apply only to more perfect twin formations, it being difficult to imagine a mere supernumerary finger or even extremity to have a similar origin.

Some have long conceived the formative power within the germ to be endowed with unusual energy, causing the development of a greater number of parts than belong to the species. The facts observed by Wolff, Von Bär, and Reichert, certainly relate to twin formations of a very early stage of development. Still this stage is too far advanced to be cited in direct support of an assumed augmentation of plastic power in the germ, as determining either an approach to the formation, out of the plastic materials of the yelk, of a double, or even of the partition of a single zona pellucida. On the other hand, these cases are of a period so early, and of characters so marked, as to render it almost self-evident that the cause of the malformation must have been a primordial one, or at least have dated from the earliest period. The multiplication of individual parts is possible at a later period, provided the germ remain for them unchanged.

2. One species of twin formations can at the present time only be satisfactorily explained by the assumption of an ovum in ovo,—one ovum being primitively enclosed within another. We refer to *twin formations from invagination or implantation*, so-termed conceptionlike germination (Meckel)—diplogénese par pénétration. One fœtus incloses, at some part, another imperfect fœtus—a fœtus in fœtu; or else one fœtus is at some one point, commonly at the skull or palate, united with another fœtus, through the medium of a more or less perfect umbilical cord. Meckel regarded the fœtus in fœtu as a product of conception, and sought to maintain this view by an appeal to analogy; adducing, for example, the formation of hair and teeth independently of copulation, sexless multiplication and propagation, regeneration. At the present day monosexual conception is hardly—multiplication by cotyledons or offshoots, in no wise—admissible. Certain of the observations in point relate to cases of malformation in the early embryo,

in the third and seventh month, for example, in which a conceptionlike product is simply impossible. The occurrence of ovum in ovo, in the instance of birds, at least, is proved; the intussusception of one ovum into another during development is, on the other hand, not conceivable.

4. Finally, an augmented number of parts depends not unfrequently upon arrest of development, and the anatomical excess is reduced to one of no real physiological import; for example, the true diverticulum of the intestine as the remnant of the omphalo-mesenteric duct, double frontal bones, and the like.

Malformations, with supernumerary parts, are divisible into several orders, which, with their principal species, are as follows:

1st order.—Malformations with individual parts supernumerary—head and trunk being single.

Dignathus. Malformation with supernumerary lower jaw.

Caudatus. Human fœtus with tail-like process at the os sacrum.

Polydactylus. Malformation with supernumerary fingers.

Notomeles. Monstrosity having supernumerary limbs at the back.

Pygomeles. Having supernumerary limbs at the os sacrum.

Gastromeles. With supernumerary limbs at the normal extremities.

To which are to be added:

1. Supernumerary skull bones.
2. „ vertebræ.
3. „ ribs.
4. „ muscles.
5. „ teeth.
6. Double tongue (always superimposed).
7. Double œsophagus.
8. True diverticulum of intestine.
9. Double cæcum and vermiform process.
10. Double pancreatic duct.
11. Double hepatic duct.
12. Manifold spleen.
13. Double heart.

14. Multiplicity of kidneys, probably due to arrest of development.
15. Double or triple ureters.
16. Double bladder.
17. Triple testicle (?).
18. Double penis and clitoris (?).
19. Double uterus (U. duplex, bicornis, bilocularis); to be regarded altogether as arrest of development.
20. Testicles and ovaries, seminal ducts, seminal vesicles, Fallopian tubes, uterus, &c., in the same individual.
21. Supernumerary mammæ.

2d order.—Twin monstrosities, with double head and trunk.

(*a*.) Double formation of the upper portions of the body.

Heteroprosopus. Two countenances; the one perfect, the other imperfect.

Dicranus. Double skull; countenance either single, or double and conjoined; lower jaw single.

Monocranus. Single skull; countenance partially double; brain double, but unequally so; three or four eyes.

Diprosopus. Double countenance; the faces and heads are completely separate, or the separation affects the faces to the zygomatic arches only; lower jaw invariably double.

Dicephalus. Two entirely separate heads, with two (seldom three) upper, and two (seldom three) lower extremities.

Thoraco-gastrodidymus. Two heads and necks, thorax and abdomen united into one; four upper and two or three lower extremities. (The Sardinian twin sisters.)

Gastrodidymus. Twins united at the lower part of the belly; the four inferior extremities branch off from the sides in pairs, at right angles.

Pygodidymus (Gurlt), *Pygopages* (G. St. Hilaire). Two completely distinct bodies, conjoined at their ossa sacra or coccygis. [The well-known Hungarian sisters, Helena and Judith, born in the year 1701, who survived their 22d year.]

(*b*.) Double formation of the nether parts of the body.

Dipygus or *Monocephalus* (Gurlt), *Thoradelphus* (G. St. Hilaire). Head, neck, and thorax single; abdomina and posterior parts separate; two or four upper, always four lower, extremities.

Heterodidymus (Gurlt), *Heteradelphus* (G. St. Hilaire); so-called parasite formation. A large, regularly formed body, bearing, at the chest or belly, another, more or less incomplete.

Dihypogastricus; so-called Janus formation. Double body, more or less coalescent above; separate from the umbilicus downwards. Here, either two heads are united with the two countenances (one of which is commonly defective), presenting in opposite directions; or else there is but a single (perhaps defective) countenance, with a double coalescent head. The trunk is double, united down to the umbilicus, and has four upper and four lower extremities.

Symphysocephalus (Barkow), *Cephalopages* (G. St. Hilaire). Twin monstrosity united at the head; the twins may be perfect, or of the one nothing may exist except the head.

(*c.*) Double formation, both above and below.

Diprosopus diædoeus (Barkow), *Tetrascelus* (Gurlt). Two heads, united at the sides; thorax and abdomen coalescent; two or four upper extremities; urinary and generative organs, as also the inferior extremities, double.

Hemipages (G. St. Hil.) The heads superficially coherent at the sides; lower jaw in common; neck, thorax, and abdomen as far down as the umbilicus, coalescent; pelves separate; four upper and four lower extremities.

Thoracodidymus (Gurlt). Two distinct bodies united at the thorax.

Xyphopages. Two perfectly distinct bodies, united only in the vicinity of the ensiform process. (The well-known Siamese twin brothers.)

3d order.—Twin malformations through implantation.

Cryptodidymus (Gurlt); so-called fœtus in fœtu. The greater, perfect fœtus bears at some point beneath the skin, or within its natural cavities, a second, smaller, and imperfect fœtus.

Omphalo-cranodidymus. The umbilical cord, together with the rudiment of the one fœtus, rooted within the skull of the other.

Epignathus. An imperfect fœtus rooted, with its blood-vessels, at the palate of a more perfect fœtus.

4th order.—*Triple monstrosities.* Their existence is confirmed by modern researches.

Supernumerary parts may be normal, both in form and structure; in both respects, however, they are frequently in various degrees defective.

The frequency of duplicate forms varies in different portions of the body; for example, a multiplication of viscera, or of organs of sense, is far more rare than of extremities.

It will be seen from the above, that in twin monstrosities the connexion between two individuals is either a mere superficial one, occurring through the medium of skin and of bone, or else one involving, at the point of union, the blending of cavities of the body, and the union, in various degrees, of the same organs in the two individuals.

Acquired preternatural increase of number consists, in man and in the higher animals, in a multiplication of the elementary constituent parts of a tissue,—of the essential or the secondary structural elements which enter into the composition of an organ. It is, therefore, the manifestation of increase of mass or density in an organ—never of the development of new, complex ones. Still, the arrest or alienation of tissues developed for the repair of injuries, or of destructive morbid processes, does sometimes determine the formation of supernumerary apparatuses foreign to the standard of the organism; for example, anomalous excretory ducts; accessory articulations.

CHAPTER II.

ANOMALIES OF SIZE.

Anomalies of volume manifest themselves as irregularities in magnitude, and as their opposite, diminutiveness, both being either congenital or acquired. They are often relative only, that is, applicable to one period of development or of life. Again, their significance and import may be limited to the proportions of the organ concerned, as in smallness of the brain; in enlargement of the heart. Finally, they refer, either uniformly or unequally, to the entire body or to individual organs.

ABNORMAL MAGNITUDE.

Congenital abnormal magnitude is sometimes general. In relation to the entire body it is termed gigantic growth—macrosomia. Some children are born inordinately large and powerful, and endowed with other marks of precocious development besides; for instance, closure of the sutures, unusual strength and length of hair, extrusion of one or more teeth. Others, impelled by innate predisposition, undergo preternatural growth during youth, and eventually arrive at dimensions exceeding the ordinary standard—in a word, grow up giants. Giant stature may depend upon the equal and proportioned lengthening of all the parts, or upon the predominant length of certain sections of the body, especially of the lower extremities. Giant stature does not needs imply corresponding development of the substance of organs and parts, certain of which may possibly have been checked in their growth; for example, the muscular system, the heart, the brain, the adipose tissue, the organs of generation.

Preternatural dimensions of individual organs of the body, both congenital and acquired, are of far more frequent occurrence. These originate in a primary anomaly or in excessive plastic activity of the germ, or, again, in hypertrophy, or in the dilatation of hollow organs, or, lastly, in a variety of diseases in which the textures of organs become involved at different periods of intra- and of extra-uterine life. These last consist for the most part, in hyperæmia, inflammation, and all kinds of heterologous formations. Congenital enlargement sometimes imports arrest of development, as, for example, a preternaturally large thymus gland.

Hypertrophy and the dilatation of hollow organs require to be considered somewhat more at large.

Hypertrophy.

Hypertrophy consists, as the term implies, in augmented nutrition, resulting in increase of mass, and generally also of volume. Long ere the term hypertrophy, and even the idea it conveys, were formally recognised in science, not only had the possibility of an increase of mass and volume without material destruction of texture been speculated upon, but the fact itself actually observed in every variety of organ. Even up to the present time, however, the recognition of this species of anomaly has been characterised by a great want of clearness and precision. It is reserved for the discrimination of the present generation, by a searching comparative enquiry, based upon a more familiar acquaintance with the normal relations of the structure and admixture of organs, and aided by the physical appliances now at our command, to make an important progressive step over this wide and fertile field.

1. *Simple augmented nutrition,* the increment of mass and of volume, not depending upon the accession of any element foreign to the organ concerned—TRUE HYPERTROPHY.

2. *Anomalous augmented nutrition.* The increase of mass and of volume is here founded upon the accession of matter alien to the organ concerned, be it formless blastema or determinate form-element. This anomalous matter, when uniformly incorporated with the texture of the organ, that is, received both betwixt and within the definite structural elements, manifests itself as *infiltration of the parenchyma*—FALSE HYPERTROPHY. It approximates closely to heterologous growth.

Such are the two sections into which, as a preliminary step, we would distinguish all the so-called hypertrophies. Each will, however, have to be specially considered in the sequel.

Widely as the *two* hypertrophies should appear to differ from each other, and little as, strictly speaking, the second series belongs hither, its consideration in this place will be found preferable as regards practical utility, and expedient for other weighty reasons.

(*a.*) From true hypertrophy to false there are insensible gradations, both qualitative and quantitative, and both forms may coexist in the same organ Thus, augmentation of the fatty contents of the hepatic cells is, by the addition of free fat and by a change in the quality of the fat, exalted into a palpable heterologous process.

(*b.*) Between the two series there exists the common connecting link that both are based upon an anomaly of the crasis; that, provided no obvious local causes prevail, both are engendered by a peculiar, personal, more or less defined, morbid tendency of general nutrition.

Every organ is by nature susceptible of, and almost every one has with more or less of precision been described as actually found affected with, hypertrophy. This does not, however, now apply equally to both categories of hypertrophy—that of the areolar and of adipose tissues, and of the muscles, more especially the organic, commonly manifesting itself as *true*, that of the so-called parenchymatous organs still more commonly as *false hypertrophy*.

(*a*.) *True hypertrophy*.

True hypertrophy appears, *à priori*, incontestable, and numberless instances have been recorded of its occurrence in every part of the body. It is remarkable, however, that when tested by an analysis, with reference to elementary texture and development, the proof is attended with extraordinary difficulty as regards the most important organs and tissues.

When it is the question, not of an obvious augmentation of the less important components of an organ—for example, of the areolar, the fibrous, the adipose tissues—but of a multiplication of the essential structural elements, the positive proof by elementary analysis often fails, although, both before and after death, the characters of the organ may seem quite sufficiently exaggerated to warrant the assumption of *true hypertrophy*.

Apart from a development of substance resulting from extraordinary succulence of the texture—that is, from its imbibing an excess of amorphous plasma more or less rich in nutrimental substances—hypertrophy can only depend either upon a multiplication of the essential textural elements by an accession of new ones, or else upon an enlargement of the original ones. Upon this point a generalisation is not feasible at the present day, and it must suffice to set forth in due succession the results of researches touching special hypertrophies.

We may specify, as unquestionable, hypertrophy of the areolar, of the fibrous, and of the adipose tissues; of the common integuments, including, not alone the cutis and the papillary bodies, but also the sebaceous glands and the epidermidal formations; of the mucous membranes and their follicles; and lastly, of the bones.

Hypertrophy cannot indeed be demonstrated by a comparative enumeration of the form-elements; and the size of the latter varies considerably even in the physiological state. Where, however, the increase of mass is obvious, and there is no accession of heterogeneous elements, the sum of the primitive form-elements must needs have become multiplied, and hypertrophy exist. We find, too, in the involved textures—for example, in areolar tissue—an extraordinary number of the elements in their embryonic stages.

Hypertrophy of muscle, however simple it may seem, is in reality most difficult of proof. The increase of mass and volume in an hypertrophied muscle certainly *seems* due to augmentation of its amount of fleshy fibres; positive evidence, however, at least with respect to the striated muscles, has hitherto been wanting. An enumeration of the primitive fibres is not feasible, nor have elements obviously engaged in the embryonic phases of new muscle-formation been as yet detected. Still less has an enlargement of the primitive muscular fibres, through increase in the amount of their primitive fibrils, been made out. That the hypertrophy consists simply in the augmented growth of the myolemma is disproved by the saturated dye, the extraordinary resiliency, the functional energy, for example, of a hypertrophied biceps brachii. Least of all could it be explained on the ground of augmented fat formation—the effect of excessive development of fat, in whatever shape, being to repel the growth of muscle. The last two propositions are moreover refuted by the hypertrophy of organic muscles.

The examination of hypertrophied hearts, for which the opportunity is frequent, offers but little assistance towards the solution of the problem, more especially where the increase of mass is considerable. A new accession of muscular fibres is not manifest. On the contrary, in proportion to the diminished energy of the organ, their fibrils are found in the progress of reduction to a partially dark coloured molecule, and of gradual extinction. One thing alone is evidently adventitious, namely, irregular aggregations of an amorphous fibro-laminated blastema, copiously interspersed with nuclei in different grades of development into areolar tissue, and of areolar tissue itself, together with a large proportion of free fat and of adipose tissue.

In the hypertrophy of organic muscle the characters are more clearly defined. Here, along with nuclei, we meet with little flat, elongated, and nucleated bodies, the rudiments of new fibres. A marked instance of hypertrophy of this nature is afforded, amongst others, by the pregnant uterus, which, at the same time, exemplifies the disintegration of fibre, and the lingering of a multiplicity of nuclei, which are themselves eventually absorbed.

Even hypertrophy of the nervous system is little more than a problem. The development of fresh nervous filaments is unproved and even improbable. Nor is the enlargement of the nerve-tubules through increase of their contents more readily demonstrable. In the central organs, and particularly in the brain, the anomaly consists in an accumulation of the minute granular connecting mass interstitial to the nerve-tubules. At the circumference it can consist only of an augmentation of the neurilemma. In the ganglia the accession of new ganglion-cells, though not ascertained, is rendered probable by the regeneration of excised ganglia.

One of the hypertrophies most frequently discussed is that of glandular bodies. We shall pass over, for the present, the false hypertrophies so frequent, particularly in the liver, the spleen, and the kidneys. That of other—for example, the mammary, the salivary—glands may consist in an augmentation either of some constituent of secondary importance—for example, areolar or adipose tissue—or of the parenchyma itself; and it is with evidence respecting this last form that we are here principally concerned.

Examined with the naked eye, the parenchyma of the enlarged prostate gland, as that best adapted for this experiment, certainly appears to have undergone an increase of mass. This might be brought about either by the creation of new acini (lobules), or by the enlargement of existing ones, through the apposition of fresh enchyma-cells; or, lastly, by the co-operation of both. The appearances in hypertrophy of the prostate gland render the new formation of lobules and of lobes highly probable. Henle's observations, however, of the existence of solitary enchyma cells in the vicinity of the glandular lobules in the lachrymal gland of a calf, and still more, what is very readily witnessed, in hypertrophied thyroid glands, render probable the new formation of such gland-cells, and, through the resorption of their partition walls, their blending with the lobules so as positively to enlarge these.

A peculiar kind of hypertrophy, concurrent with dilatation of the cavities of the acini, is a very frequent cause of the enlargement of glandular formations. This dilatation is due to an augmented secretion taking place within the follicles, determined by the same local or general causes as the hypertrophy itself; and this latter consists in an increase of mass in the investing fibres of the follicles. This condition is immediately followed by the expansion or degeneration of the follicle to a dilated cyst-like cell, with a stouter lamina of enveloping fibres. At the same time the secretion may become alienated both in quantity and in quality, until a cyst is completed with contents altogether alien to the native secretion of the gland. This is witnessed in the follicles of the thyroid gland, in the Malpighian bodies of the kidneys, in the Graafian follicles, and in the acini of the salivary glands; in the mucous follicles, particularly those of the cervix uteri, where, even in the physiological state, they frequently dilate into capacious thick-coated cysts, rupture, and discharge their contents.

Evidence of hypertrophy of the liver, of the spleen, of the lymphatic glands, is hardly obtainable.

The idea of a hypertrophy of the liver from the accessory formation of new hepatic cells would not indeed be discordant with our notions of the functional importance of that organ. All anatomical proof is, however, unattainable. That, on the other hand, upon which it more *obviously* depends, is turgescence of the hepatic cells from an increased proportion of fat and of bile, together with hyperæmia of the capillaries. This condition determines the more or less marked development of what is called the secreting substance of the liver,—one-sided hypertrophy of the liver, as it is termed, or nutmeg liver.

Hypertrophy of the spleen must be referred, first, indeed, to the reinforcement of its fibrous framework, but mainly to augmentation of the pulpy parenchyma of the spleen, that is, of the elements out of which it is constructed.

Nor can hypertrophy of the lymphatic glands be well traced to the adventitious development of new lymphatic vessels between their parenchyma, but rather to increase of the parenchyma between the lymphatic vessels. It is certain at least, that in atrophy, the lymphatic vessels become deficient in parenchyma.

Hypertrophy of the lungs consists not in the addition of new cells, but in an augmentation of matter in the parietes of the existing ones. The ample, energetic (vicariating) function, moreover, of a hypertrophied lung seems to imply a multiplication of the capillary vessels by the creation of new ones. Thus, again, hypertrophy of the corpora cavernosa does not depend upon the addition of new cellular spaces, or their increase through the development of new septa, but upon increase of substance, thickening of the walls of the cellular spaces, with simultaneous dilatation of these latter.

(*b.*) *False hypertrophy.*

This has been already adverted to as a heterologous product. As such it occurs frequently in the form of infiltration. False hypertrophy is for the most part cognisable at a glance from the alienation which the general characters of the organ have undergone. Very marked hypertrophies of this kind are found to affect the liver, and with rather less frequency the spleen and even the kidneys; presenting, in the instance of the two former, what is commonly termed hypertrophy, physconia, engorgement, &c. These manifest themselves in the shape of fatty liver, waxy liver, of albuminous, lardaceous infiltration of that viscus, of the spleen, of the kidneys, and they will be reconsidered under the head of heterologous growths. They not rarely attain to a very high grade, are always distinguished as being palpably based in a constitutional dyscrasis, and are, proportionately to the rapidity or slowness of their development, attended or unattended with pain.

To these hypertrophies, moreover, properly belongs the ultimate degeneration of hypertrophied and dilated glandular follicles into cysts.

Finally, we may here class all hypertrophies founded upon products of inflammation, so far as they consist in the adventitious development of a blastema foreign to the texture involved, and convertible into areolar and fibroid tissue.

Hypertrophy attacks one, or a few disconnected, but for the most part nearly kindred organs; or, again, an entire system—for example, the osseous, the lymphatic system. The general hypertrophy called polysarcia or corpulence, consists both in the excessive development of fat, and in extraordinary succulence of the soft tissues, more especially of the areolar.

Hypertrophied organs offer a variety of remarkable changes. The volume is usually augmented,—more obviously so in the case of false hypertrophies. Now and then the natural volume is retained, the failure of increase of volume being compensated for by augmented density of the organ, or some one anatomical constituent becoming hypertrophied at the expense of another one, which wastes in a corresponding degree. In hollow organs we distinguish between a *simple* hypertrophy with normal capacity, an *excentrical* with dilatation, and a *concentrical* with diminution of the cavity. In this last the volume of the organ may be augmented, or normal, or even diminished. Examples are afforded in hypertrophies of the heart, of the uterus, &c.

The weight of hypertrophied organs corresponds with their increase of volume and of density.

The shape always undergoes a change proportionate to the degree of the enlargement. Generally speaking, hypertrophied organs assume a certain roundness, losing their edges, their angles, and their flat surfaces. In the case of some organs, and of the liver in particular, the marked character of such disfigurement is not devoid of pathognomonic significance.

The *colour* is, in *true* hypertrophy, the normal—only of deeper tint. Take, for example, the saturated red in true hypertrophy of muscular flesh, the saturated twofold coloration of nutmeg liver. In false hypertrophy, the coloration suffers various alterations.

The consistence of an hypertrophied organ is often unchanged, often increased, sometimes diminished. A remarkable degree of density and of resiliency characterises hypertrophied muscle, more especially in the right ventricle of the heart; and, again, in hypertrophy of the spleen referrible to mechanical hyperæmia, and unattended by obvious enlargement. The same observation applies to false hypertrophies, in particular to brawn-like infiltration of the liver, the spleen, and the kidneys. *Fatty* degeneration of the liver is marked by a diminution of consistence.

The blood-vessels of hypertrophied organs sometimes present a dilated calibre and thickened (hypertrophied) coats. This is especially perceptible in congestion and hypertrophy of long standing; not so, or at least not in a marked degree, in other cases. Does the accessory formation of new structural elements in the hypertrophied organ imply that of new blood-vessels likewise? Direct experience affords no information upon this point. In relation to the hypertrophy of vicariating organs, and of the lungs more particularly, it would be reasonable, where vicarious action really is in force, to take for granted the accessory formation of new capillaries. The nerves of hypertrophied organs are occasionally found considerably thicker than natural.

The causes of hypertrophy are:

1. Morbid increase of the quantity of blood in the capillaries of, and retarded circulation in, the affected organ; repeated and abiding hyperæmia. Examples are furnished in particular by the frequent hypertrophies of the abdominal viscera arising out of mechanical hyperæmia, of the mucous membranes in organic diseases of the heart, of the areolar tissue in the lower extremities in a varicose condition of their veins, and, lastly, by the hypertrophies of the mucous membranes brought about by the hyperæmia entailed by repeated inflammation.

2. Augmented, violent action induced by various direct or reflected stimuli. Examples present themselves in hypertrophy of the voluntary muscles, of the heart, of the organic fleshy tunics.

3. The groundwork of a lengthy series of hypertrophies consists in *a constitutional vice of nutrition and in an anomalous blood-crasis.* The hypertrophy is here the expression, the symptom, of general impairment. This applies to true, and with greater force to false hypertrophy. To this class belong hyperostosis, excessive development of fat, endemic goître,—hypertrophy of the brain, and hypertrophy of the lymphatic glands in rhachitism,—the excessive development of fat with simultaneous impairment of its quality in alcohol-dyscrasis,—the conditions of fatty, of waxy liver, of brawny infiltration of this organ, of the spleen, of the kidneys in tuberculosis, rhachitisms, inveterate syphilis, &c.

4. *Inflammation;* the result of which is so-called *inflammatory*

hypertrophy, to which we shall have to recur by-and-by. It engenders *true* hypertrophy in areolar and osseous textures alone; in all the rest, through the fresh deposition of areolar and of fibroid tissues, *false* hypertrophy.

Hypertrophies of both kinds are either congenital, or, what is far more frequent, acquired during extra-uterine life.

The *course* of hypertrophies is for the most part chronic. Nevertheless, they not rarely form within a surprisingly short period, or from time to time rapidly increase. They are then often painful affections, as, for example, the acutely developed fatty liver.

Hypertrophy, when it has attained a high degree, impairs the function of the affected organ, whilst the latter, by its increase of weight and of volume, obstructs the function of neighbouring parts.

Of itself it commonly proves fatal through palsy, the result of the ultimate disproportion between the bulk of the hypertrophied organ and the powers of innervation. As examples, may be cited hypertrophies of the heart, palsy of the hypertrophied intestine above a stricture, palsy of the hypertrophied urinary bladder, and the like.

A proper discrimination is requisite between increase of volume from hypertrophy and the dilatation of hollow organs, more especially if associated with attenuation of the parietes. Dilatation is generally coupled with hypertrophy of the walls of the dilated organ,—termed *active dilatation,* co-significant with *excentrical hypertrophy. Simple dilatation,* in which the walls are of their natural thickness, is a kindred form. Dilatation may, however, be conjoined with attenuation of the walls; it is then denominated *passive dilatation.*

The causes of the dilatation of hollow organs are various.

1. *Mechanical impediments,* which obstruct the free passage and egression of the contents of the different canals and reservoirs. They occasion dilatation either beyond or behind their seat, and manifest themselves—

(*a.*) As local constriction of calibre, through pressure from without.

(*b.*) As coarctation consequent upon hypertrophy and change of texture in the walls of the organ. In instances rare, except in disease of the heart, as dilatation. Thus, whereas in the intestinal canal it is the accumulation of its contents, on the other hand, in dilatation of the orifices of the heart it is the increased diameter of the blood-column, in insufficiency of the heart-valves the regurgitation of the blood, that furnishes the mechanical impediment.

(*c.*) As obturation of canals with substances of various kinds, whether introduced from without or begotten within the organism, whether closing up by their bulk or obstructing by their aggregation,—in a word, as foreign bodies, secretions, &c.

Other local causes, however, besides the above-mentioned—accumulations of foreign bodies, of self-engendered deposits—are in like manner productive of dilatation.

2. Paralysis of the contractile elements in the walls of the organ, whether peripherous, and consecutive to mechanical, concussive violence, tension, &c., to disease of texture, especially inflammation; or determined by affection of the nervous centres.

3. Diseases of texture; for example, fatty degeneration, particularly of the heart.

4. *Inflammations.*

The different causes frequently act in unison in various sequences. Thus, coarctation begets accumulation of contents. This, together with a certain relative amount of existing dilatation, occasions paralysis of the organ. The paralysis causes dilatation, and thereby accumulation of the contents, which again, in turn, mechanically promotes the dilatation.

Dilatation destroys life through paralysis, either simply or with the concurrence of asthenic stasis, inflammation, and gangrene, towards the establishment of which the contact of retained contents in the progress of decomposition contributes its part. Take for example the intestinal canal, the urinary bladder, &c.

Sudden dilatation is wont to assume the passive character, a supervenient hypertrophy being more marked in the inverse ratio of the celerity with which the dilatation is brought about.

Increase of volume in one direction at the expense of the general bulk of the organ, the result of forcible tension, is distinct from hypertrophy.

ABNORMAL DIMINUTIVENESS.

Congenital abnormal diminutiveness affects the entire body, as *dwarf stature* (microsomia), the individuals being termed dwarfs, or pigmies. These are either born diminutive, or, owing to inherent predisposition, not developed after birth to the ordinary stature. Dwarf-growth manifests itself either in the corporeal development remaining stationary at the stage of childhood, the not unpleasing outlines and proportions of which it then retains, or else it is founded in an arrest in the growth of the bones, especially those of the lower extremities, with simultaneous malformation of the osseous trunk. It is marked by a disproportion in the more important parts of the body—largeness and hydrocephalic shape of the skull; length of trunk coupled with shortness of extremities, especially of the inferior ones; deformity of bones, consisting in thickness, especially of the articular terminations. This latter dwarf-formation is always congenital, and the bone affection upon which it depends has been designated as congenital rhachitis; against which, however, we have as yet to urge that, however much its features resemble those of the rickets of childhood, the direct evidence of its identity with the latter is wanting.

Accordingly, dwarf-growths may depend, either upon a primitive vice of plasticity, or in an anomaly of development affecting specifically the osseous system. Growth may, moreover, become checked at an earlier or later period, subsequent to birth, by constitutional maladies of an exhausting kind, both congenital and acquired, and especially by such as affect the brain or spinal medulla.

Partial diminutiveness affects individual organs, systems, or sections of the body. It is founded sometimes in a primitive anomaly of, or in defective plasticity in, the germ; in pressure and in restriction of space within the uterus; or, again, in a hindrance to growth after birth, resulting from exhausting diseases, from paralysis; lastly, in atrophy. Where larger sections of the body are affected, such disproportions result as are observed in giant-growth, in dwarf-growth, and in numerous descriptions of monstrosity. Although it may affect every organ and system, it is nevertheless most conspicuous in the following: namely, the brain (and skull) [microcephalia];

the eyes [microphthalmus], the inferior maxilla [brachygnathus], the lungs (and thorax), the stomach and intestinal canal, the common integuments (shortness), the muscles, the skeleton, the heart and vascular system (especially the aortal), the generative apparatus.

It is necessary to observe, that—

(*a.*) In monstrosities, accessory, supernumerary parts are very frequently diminutive.

(*b.*) Formations checked in the development of their mass and volume, often exhibit an arrest in the development of their texture; for example, bones, muscles.

(*c.*) Preternaturally diminutive organs often display some other kind of deformity, referable to the same causal relations.

(*d.*) Next to diminutiveness is total absence, which, in reality, often applies to individual formations entering into the composition of a complex part or system.

In hollow organs preternatural diminutiveness manifests itself as coarctation, and even as complete imperviousness, which, when affecting the external orifice of canals, is termed imperforatio, atresia.

As contrasting with hypertrophy, atrophy here demands a special consideration.

Atrophy.

Atrophy, wasting [tabes], consists in the withdrawal from a formation, after it has reached a certain grade of maturity and bulk, of its constituent elements, without any compensating regeneration of these; the result being decrease of substance, usually coupled with diminution of volume.

General atrophy attacks simultaneously, or in rapid succession, many organs and systems, if not all. *Partial atrophy,* one organ exclusively, or at least preferably. To the latter we shall at once direct our attention.

As with hypertrophy, so every organ is liable to become affected with atrophy.

Atrophy may, in the first place, be essentially primary, that is, developed in an organ as its first and sole anomaly, through influences more or less palpable, but external to such organ. Or, again, it may be secondary, that is, the result of previous textural alteration in the organ. The first is akin and analogous to the periodical intra- and extra-uterine processes of involution of certain formations, as also to the senile atrophy or *marasmus* of organs.

Primary partial atrophy is often, indeed, purely local; in not a few instances, however, it is probably conditional upon a general derangement of nutrition, of which, in such case, it is but the manifestation or symptom.

Causes of partial atrophy are:

1. *Diminished supply of blood*—of alimentary fluid—owing to compression, obturation, coarctation, or obliteration of the afferent blood-vessels; for example, partial atrophy with lobulation of the liver from adhesive phlebitis of branches of the portal vein, atrophy of the cartilaginous investments of the joints from sclerosis of the spongy condyles, and the like. Thickening of the minute and capillary vessels from within may co-operate with ossification of the great arteries in producing atrophy, especially in the brain, by rendering the walls of such vessels impermeable to the plasma of the blood.

2. *Exhausting disease, or healing process;* for example, atrophy of the bones and of their adjacent soft parts as a consequence of caries, of destructive suppuration in the effort to repair injuries; atrophy of the uterus after childbed, and exhausting puerperal diseases, &c.

3. *Diminished innervation,* paralysis, or impeded action of an organ owing to mischief of a mechanical nature; for example, atrophy of muscles in anchylosis, in luxations.

4. *Pressure and distension.*—These occasion increased absorption, a species of atrophy designated by the term *detritus, usura.* Even the most stubborn textures are not proof against it, the rigid osseous texture itself being in a high degree susceptible of it. It not unfrequently advances to the degree of a lesion of continuity.

5. *Anomalies affecting general nutrition, and the blood-crasis in particular.* Upon such are based, for example, perhaps, the untimely decline (involution) of the generative organs in either, but especially in the male sex; but with more of certainty, several painful kinds of atrophy of the osseous system. Acute yellow atrophy of the liver is unquestionably founded upon an

anomaly of the crasis, whilst the thyroid gland is atrophied by the fluids becoming impregnated with iodine.

6. *Consecutive atrophy* depends, as already stated, upon a previous alteration of texture, a breaking up thereof through hæmorrhage (apoplexy), inflammation, and heterologous growths. Two contingencies may here arise; either the adventitious product and also the disabled textural elements of the organ may both undergo absorption, or else these latter may waste away alone, leaving in their place the new product, in the original or in subsequently diminished proportion and indefinite shape. Exemplifications occur in cell-infiltration of the medullary substance of the brain consequent upon encephalitis; in atrophy of the kidneys, resulting from inflammation or from Bright's granular disease; finally, in the merging of muscular fibre in the fatty degeneration of muscle and of normal textures in heterologous growths.

The morphological process connected with atrophy is not known in detail. To judge by a few facts—for example, the reduction of the uterus after delivery, the perishing of muscular fibre in fatty degeneration, the wasting of nerves, of lymphatic glands—the process essentially consists in the breaking down and liquefaction of the secondary elements (fibre), resulting from the metamorphosis of the cells and from the cells themselves. The nuclei at first remain, but subsequently undergo the same reduction and ultimate resorption. In the case of new growths, this blastema, arising out of the wreck of the said elementary bodies, may become subservient to the construction of anomalous textures.

With respect to the changes suffered by atrophied organs in their physical properties, we may offer the following general remarks.

The volume of the atrophied organ is indeed very commonly diminished, membranous formations having become thinner: this is, however, by no means invariably or immediately the case, at least not in any marked degree; for example, in atrophy of the lungs or of the bones. In hollow organs the volume may, owing to a coexistent passive dilatation, even become augmented. In such organs atrophy is conjoined either with normal capacity, *simple atrophy;* or else with dilatation of the cavity, so-called *eccentrical atrophy*—for example, of the heart, of the uterus, &c.; or, lastly, with coarctation, *concentrical atrophy,* in which the depth of the walls may be natural, or even greater than natural.

The weight of atrophied organs may be reduced, normal, or even increased: in the first case, proportionately to the simplicity of the atrophy; in the two latter cases the atrophy is consecutive, new growths supplanting the original textures.

The shape of atrophied organs embraces a variety of anomalies; amongst which we may specify the deformity which attaches to the concentrical wasting of hollow organs and organs of cellular structure like the bones, the removal of the incisura interlobularis in atrophy of the lungs, the tuberous gland-like surface in secondary atrophy of the kidneys, &c.

The structure of atrophied organs involves various, and occasionally very marked changes. Thus, organs of cellular, of cavernous structure, by dint of the absorption which takes place at the parietes of their cells and canals, are rendered wide-celled—for example, in the lungs and in bones; and this structure eventually dwindles into a mere net or trellis work. In consecutive atrophy, a new growth of a completely different texture occupies the place of the original structure; after inflammation, for instance, a honey-combed, meshy, or, on the contrary, a dense, callous, areolar tissue.

The consistence is in like manner subject to many changes. It is sometimes diminished, readily giving rise, upon slight occasion, to lesions of continuity; in the osseous system, for example. Sometimes it is increased. Secondary atrophy presents, in a marked degree, either contingency, according to the particular change of structure. Acute processes of the reduction of mass and volume determine, in certain organs, rather a decrease—chronic reduction rather an increase—of consistence.

Atrophied organs have a tendency to paleness of colour. This, however, in some measure accords with certain changes affecting the native pigment of organs; for example, the decoloration of muscle, of the spleen to rust-brown, fawn-colour, or yeast-colour. The rule itself is, moreover, subject to sundry exceptions. Thus, the pure white of the medullary substance of the brain is exchanged for a whitish-brown. Organs that become atrophied without a proportionate thinning of their capillaries, sometimes, by dint of a *relatively* augmented supply of blood, assume a deeper colour; for example, bones, kidneys. At the same time much depends upon the character of the atrophy; as in the cases of red and yellow atrophy of the liver.

The blood-vessels of atrophied organs become reduced in calibre, collapsed, and finally cut off from the atrophied organ, that is, the connexion interrupted between its obliterated capillaries and the vascular trunk. This, however, has, in like manner, its exceptions, as in the case of dilatation of the trunk and ramifications of the pulmonary artery in atrophy and in emphysema of the lungs; in the case of dilatation of the blood-vessels of the brain, in atrophy of this organ.

The nerves of atrophied organs in all probability dwindle *pari passu* with the wasting of the diseased textures.

Atrophy is sometimes an acute, but more commonly a chronic process. In the former case it is frequently a painful affection.

The consequences of partial atrophy differ vastly in different organs. Either they are limited to a small range, to the locality itself, or they implicate more or less sensibly the entire organism. In this respect, atrophy of the central organs of the nervous system, and of the organs presiding over the preparation of the blood and over the grand secretory functions, namely, of the lungs, liver, and kidneys, is of course foremost in importance.

General atrophy in the form of emaciation, consumption, affects, indeed, the entire body, but by no means all organs and systems simultaneously or in an equal degree. Next in order to the falling off in the amount of blood, is that of the adipose, the areolar, and kindred tissues; then follows that of the voluntary muscles, then of the organic fleshy tunics and of parenchymatous organs, lastly of bones; whilst, even in the highest grades of the affection, the nervous system, so far as relates to its constituent elements, remains exempt. This order is, however, subject to many exceptions. The wasting of several formations low in the scale above laid down, occurs primitively, and offers the starting point for the atrophy of the rest; for example, atrophy of bone.

The causes are loss of fluids of whatever kind, deficient reproduction of organic substance, fasting, various affections of the digestive organs, bodily and mental exertion, inordinate activity of the nervous system in various ways, excessive heterologous development, dyscrasis of the blood.

It is not rarely combined with the hypertrophy—for the most part, false hypertrophy—of internal organs, especially of the liver, spleen, and lymphatic glands.

A peculiar form of partial diminution is represented in the *coarctation of canals and cavities.* It is often, indeed, essentially a concentrical atrophy. It may, however, arise from external pressure, from deficiency of contents, from continued irritation of the sensitive parietes, or even from hypertrophy of,—or from various heterologous luxuriations and changes of texture implicating,—the said parietes. The highest grade manifests itself as morbid closure (atresia).

CHAPTER III.

ANOMALIES OF FORM.

Anomaly of form, or *deformity,* affects either the entire body or portions of it only,—*general* or *partial deformity.* It is either *primitive* or *acquired; simple* or *complicated,* that is, conjoined with anomalies of a different nature.

General deformity is rare, even as relates to very faulty abortions.

To *primitive, simple, partial deformities* belong—

(*a.*) Those in which any part is preternaturally long, broad, thick, spherical, angular, curved, &c.; for example, oval, vertical pupil, oblique uterus.

(*b.*) The division of parenchymatous organs into two or more parts, by extraordinary lobulation (the lungs, liver, spleen, kidneys); the section of hollow organs by the inordinately sharp partition of a naturally double cavity, or by septformation in a cavity normally single; for example, double apex of the heart, bilocular uterus.

Many of the former, and still more of the latter kinds bear the impress of arrest of development, and present the images of brutes.

Amongst *primitive complicated deformities* may be classed most of the instances of disproportion and of absence of symmetry manifested in the preternatural volume of individual organs or sections of organs; secondly, those consisting in abnormal position, abnormal association, coalition or cleft-formation; and, lastly, those founded in the superfluity or in the deficiency of parts.

Pre-eminent amongst them are hermaphrodites. The forms of hermaphroditism, strictly considered, range under several of the heads just specified as conditional upon anomalies of shape. It would, however, appear most suitable to discuss them here under a single head, seeing that from one or more fundamental anomalies inductive of hermaphroditism, there often results a marked deformity of the generative organs; seeing also that the character of many of them consists *essentially* in a departure from the normal type. They are, in the great majority of cases, arrests of development.

In strict analogy with the relations of lower orders of animals, those malformations should be designated as hermaphrodites, in which the generative organs of both sexes are found united in a single individual. Such monstrosities have, from time immemorial, been abundantly described. We must, however, unite with Joh. Müller and Th. Bischoff in rejecting the great majority of these examples. Bischoff has pointed out the numerous sources of error by which, in such cases, a judgment may be warped; as, for instance, the great resemblance between the generative organs of the two sexes at an early period, the uniform type in the development of both, the coalition of the corpora Wolffiana, the errors formerly prevalent as to the primitive identity of both sexes. It is, therefore, easily intelligible that a judgment to be relied upon can alone be based upon a familiar knowledge of the progressive development of the genital organs, and of their elementary structure. The coexistence of testicles and of ovaries on the same side has been thrown into entire discredit by the arguments of Joh. Müller, who nevertheless admits the occurrence of ovaries on one side and of testicles on the other. Th. Bischoff, however, impugns the accuracy even of the latter observation. Nor will Bischoff unconditionally admit the numerous cases of other portions of the genital organs alleged to have been found bisexual on the same side, or male on the one side and female on the other. The history of development, he affirms, sufficiently teaches us that this species of simulation may be the result partly of an arrest, partly of a peculiar modification in the type of development. Moreover, the progressive development of the uterus, of the seminal vesicles, of the prostate gland, and of Cowper's glands, in both sexes, still remains, notwithstanding the skilful investigations of J. Müller, Rathke, Valentin, so far matter of uncertainty that we can hardly derive any support from analogy with the normal state.

Strictly speaking, therefore, neither in man nor in the higher animals, can hermaphroditism, that is, the coexistence of testicles with ovaries, occur. So far as relates to these essential organs of generation, there can be but male or but female individuals. On the other hand, the rest of the genital organs, which in their rudimental condition closely resemble each other in the two sexes, may, owing to some anomaly in the mode of their development, assume in a male individual more or less of the feminine, in a female individual more or less of the masculine form—and thus, in either case, the semblance of both combined.

If, with Bischoff, we rightly discard from hermaphroditism, cases of individuals with throughout female organs but masculine habit, and, again, with perfect male organs and feminine habit—irrespectively of a simply undersized penis or a preternaturally developed clitoris—we may, consistently with our usual classification, divide hermaphrodites into—

1. Those which being, as to the essential organs of generation (testicles and ovaries), distinctly male or female, exhibit nevertheless some anomaly of development [be it arrest, overgrowth (up to the masculine type), or disproportion of some other kind] more or less typical of the opposite sex.

(*a.*) *Hypospadia* in its higher grades, namely, on the one side with cleft scrotum and the formation of a vagina-like sinus—on the other side, as its analogue, diminutive vagina, closure thereof into a raphe or suture, partial or entire absence of this organ, with a clitoris developed into the semblance of a penis hypospadiæus, or one completely channelled with a urethra.

(*b.*) *Cryptorchism:* concealed testicles in the one case; in the other its parallel condition, descent of the ovaries into the greater labia pudendi. Now and then associated with the foregoing form.

High grades of these anomalies constitute the so-called transverse hermaphroditism, implying external organs of the one and internal of the other sex. The case of externally female and internally male organs is by far the more common, because due to an arrest in the development of the male organs, whilst the opposite case depends upon the ulterior development of the female organs into the male type.

(*c*.) The occurrence in the male sex of a womb-like organ.

These cases collectively constitute what is termed spurious hermaphroditism.

2. *Lateral hermaphroditism.* The presence of testicles and vas deferens, with or without seminal vesicles, on one side, and of ovarium and tube on the other. It has been before stated that Bischoff attaches little credit to these alleged cases of the coexistence of testicle with ovary.

3. True hermaphroditism (hermaphrodite per excessum, androgynus, coexistence of male and of female organs on the same side). With reference to these cases, recorded by Meckel and by Gurlt, Bischoff remarks that not a single one offers conclusive evidence of the union of the two main organs of generation, the testicle and ovary, and that the seeming dualism of the rest of the organs is explicable according to principles of normal development.

Amongst *acquired deviations* of form are to be enumerated, first, those conditional upon hypertrophy and atrophy; upon change of locality and of connexion—as, for instance, hernia, prolapse, oblique position of the uterus from one-sided traction, luxation; upon mechanical interference—for example, amputation, extirpation; upon cicatrisation; and, lastly, those malformations of organs which essentially depend upon alterations of texture—misshapen liver, for instance.

The most frequent and marked kinds of deformity are founded upon anomalies of the osseous system; for instance, curvatures of the spine, of the long cylindrical bones, dislocations, preternatural articulations, &c.

CHAPTER IV.

ANOMALIES OF POSITION.

Preternatural position—situs mutatus, inversus, alienus, dislocatio, ectopia—is either congenital or acquired. In either case, it may affect a single organ or implicate several.

To congenital anomalies of the kind belong:

1. The re-establishment of symmetry, in lateral asymmetria. For example, each lung is found to have two lobes only, with both liver and heart in the centre. This is probably an arrest of development, these organs originally occupying the median line, and being in appearance symmetrically constituted.

2. *Lateral transposition, displacement from side to side,* affects either only individual organs of the thoracic or abdominal cavities—the cæcum being, for instance, on the left, the heart on the right side; or else it affects the aggregate of the thoracic or of the abdominal viscera; or, lastly, and most commonly, the collective organs of both these cavities at once. The type of formation is reversed, the right greater lobe of the liver, for instance, becoming the left, the left becoming the right, the gall-bladder lying to the left of the longitudinal fissure. As regards the cause, it appears to Bischoff that in the embryo at an early period, the umbilical vesicle, after development of the intestine, verges towards the left, and the allantois towards the right, whereby a peculiar spiral revolution of the embryo is effected, which may possibly influence the position of the internal organs. It is conceivable that a change in the position of the germinal vesicle in the ovum might, in like manner, give rise to a transposition of organs.

3. *Transposition from above and below.* Thoracic organs in the abdomen; abdominal in the thorax.

4. *Transposition from front to back;* for instance, in the case of teeth, in distortion of the extremities.

5. *Displacement of individual organs from the median line,* as, for instance, of the falx (cerebri),—of the uterus. *Displacement upwards,* as in cervical position of the heart. *Displacement downwards,* as in abdominal site of the heart, pelvic position of the kidneys.

Anomalous origin and distribution of arteries and veins. The more important examples hereof will be discussed under the head of special anomalies of the heart. They originate, for the most part, from blood-vessels which should have become further developed stopping short in their progress, whilst others which should have remained diminutive, or even have disappeared, persist and become more strongly developed. The majority represent types proper to different vertebrata—to fishes, amphibia, birds, and mammalia.

The preternatural position of certain organs which, in their development, undergo locomotion to a considerable extent, is specially termed *deviation, aberration.* An example offers in the descent of the testicle beneath the femoral arch or into the perinæum. In truth, many anomalies of position are founded in an early aberration in this sense. The same designation is applied to anomalies in the origin, course, and ramification of vessels.

Acquired transposition is of various kinds, and many of these so closely resemble the congenital forms, as with difficulty to be distinguished from them. Their import varies greatly, proportionally to—

(*a.*) The importance of the organ displaced;

(*b.*) The number of organs displaced;

(*c.*) The extent of the displacement; and

(*d.*) Especially to the rapidity with which the dislodgment takes place, and to the corresponding strain upon various formations, more particularly blood-vessels and nerves.

(*e.*) The extent of the morbid complication to which the displacement is due; for example, mechanical injury to the surrounding parts.

(*f.*) The degree of embarrassment to which the dislodged organs become subject; for instance, limitation of space, incarceration, exposure to the external air, &c.

(*g.*) The amount of functional embarrassment inflicted upon organs by the displaced parts; for example, upon the lungs by the intrusion of abdominal viscera into the thoracic cavity.

These transpositions are, moreover, *spontaneous,* where the organ changes its position owing to increase of volume, of mass, or of weight, in which case it commonly sinks into a lower region. Or else they depend upon conditions extraneous to the organ displaced; to which class belong dislodgments consequent upon atony of investing, supporting, attaching formations, especially when of a muscular and fibrous nature. Or they are referable to tonic spasm and retraction of fleshy, of tendinous, and of ligamentous formations, as exemplified in hernia, in curvature and distortion of the spine, in luxations, in club-foot, &c. Lastly, we have to mention the displacement of organs through tumours, through dislodged or enlarged neighbouring organs, through accumulated fluids, and the like.

The more important forms of displacement are:

1. *Hernia*; the extrusion of one or more viscera, or of merely a portion of a viscus, out of its natural cavity into a sac formed by the circumscribed dilatation of the membranous investments of that cavity (hernial sac).

2. *Prolapsus*; the naked extrusion of a viscus through a natural orifice. It is either complete or only partial; the former case occurs in hollow organs—for example, in the rectum, in the prolapsed and inverted womb. At an external opening of the body intussusception becomes prolapsus, which is intussusception minus the external layer or sheath.

3. *Protrusion*, propendentia, of viscera, owing to congenital fissure, or to rupture or penetrating wounds of the parietes of cavities.

Again, the position of organs may be anomalous, independently of any change of place, simply by preternatural inclination, especially in the shape of obliquity. This species of deviation is sometimes primitive and congenital, sometimes acquired. It affects the eye, the heart, the stomach, the uterus, the teeth, &c. It is frequently coupled with obliquity of form, as in the case of the uterus.

CHAPTER V.

ANOMALIES OF CONNEXION.

These anomalies (vitia nexûs) consist in diminution or total absence, or else in enhancement of the natural connexion and contiguity of organs. They are both primitive and acquired, and, in either case, exceedingly various in degree and extent. To the former belong the opposite extremes of cleft-formation, and of malformation from fusion, together with atresia.

1. Cleft-formations.

(*a.*) A considerable number of these have their foundation in the germ being originally a membranous expansion, the edges of which incline towards each other, eventually meet and thus form into cavities or cylinders.

The two cavities developed out of the animal layer of the germ, for the inclosure of the central nervous system and of the organs of the neck, the thorax, and the abdomen, are formed out of the union of the so-termed abdominal and visceral plates. Now, supposing the union of the edges of these plates not to take place at all, or to take place but imperfectly; or supposing consummated union to become re-dissolved through some agency, like the accumulation of watery fluid; there would result, either anteriorly or posteriorly, and commonly at the median line, although often elsewhere, a cleft or gap, attended by prolapse, or even by destruction of the implicated viscera.

Clefts of this kind are:

Cleft skull (hemicephalia).
Cleft spine (spina bifida).
Cleft countenance.
Cleft cheek.
Cleft palate.
Cleft upper-lip.
Cleft tongue.
Cleft in the thorax.
Cleft in the abdomen.
Cleft in the pelvis.
Cleft urinary bladder, so termed prolapsus, inversio vesicæ.
Cleft dorsum penis (epispadiasis).

The last two are generally combined with cleft pelvis.

The intestinal canal is in like manner developed out of an expansive formation, the united vascular and vegetative layers of the germ, by the approximation of its edges, out of a groove in front of the vertebral column. Hence, clefts occur in the intestinal canal, in the stomach, as arrested growths.

(*b.*) Other clefts, besides those mentioned, originate in the gaps which occur during the normal development of particular parts, not closed at the proper time. To these belong:

Cleft choroid membrane and iris (*coloboma iridis*). In the embryos of all vertebrata we meet, at an early period, at the inner, lower angle of the eye, with a narrow colourless stripe in the choroid membrane, which commonly disappears before the iris becomes developed. When this stripe continues beyond this period, it often abides in the iris, and is perceptible after birth.

Cleft at the side of the neck, congenital fistula of the neck, founded in the mode of development of the visceral cavity of the head. The visceral edges of the animal layer of the germ do not grow towards each other in continuity, but in ridges termed visceral or branchial arches, which are parted by fissures termed visceral or branchial clefts. When the early closure of these does not take place, occasion is given to the somewhat rare malformation in question.

Cleft urethra and scrotum (*hypospadiasis*) of various grades. At an early period is discoverable, at the lower side of the rudiment of the penis, a groove, which extends to the common orifice of the urinary and sexual organs. In the male, the edges of this groove being brought into apposition, coalesce into a raphe or suture, and thus form at once the scrotum and the urethra. Where this process wholly or partially fails, there arises a malformation which, if the penis be at the same time short and the testicles retained within the abdomen, simulates female development—a form of spurious hermaphroditism.

To this malformation succeeds—

Cloacal formation, junction of the orifice of the anus and of the external orifice of the urinary and sexual organs—a formation which, being at an early period normal, may, through an arrest of development, become persistent. In the male it is necessarily associated with the last-mentioned vice of formation, that is, with hypospadiasis, frequently also with cryptorchism.

(*c.*) As cleft-formations may likewise be reckoned the persistence of certain communicating apertures between parts which, at a later period, ought to remain separate, as also the abiding patency of certain canals, namely:

Defective development of the septa of the heart's ventricles and auricles; permanent patency of the foramen ovale. These septa form only gradually within the heart, the septum of the auricles not arriving at its full development until after birth. Defective development of the septum of the ventricles occasions a resemblance with the hearts of fishes and of reptiles (the crocodile excepted), and especially of serpents and tortoises; absence of the septum of the auricles a resemblance, in particular, with the hearts of fishes. It is often quite evident that the arrest of development has been caused by endocarditic changes in the valves—the residue of fœtal valvular inflammation.

Abiding patency of the ductus arteriosus, ulterior dilatation thereof.

Abiding patency of the ductus venosus Arantii, giving rise to the abduction of a portion of blood from the vena portæ into the vena cava.

Abiding patency of the processus vaginalis peritonæi (the upper portion of the tunica vaginalis testis), so commonly the cause of congenital hernia or hydrocele. Generally speaking, the inguinal canal closes immediately after the testis has, in the seventh month, descended into the scrotal sac, carrying with it a process or continuation of the peritonæum. Occasionally an arrest of development prevents the said closure from taking place.

Abiding patency of the urachus, allowing the escape of urine through the umbilicus. Urachus and urinary bladder are the portions of the allantois internal to the embryo, which is destined to convey the umbilical vessels from the embryo to the external membrane of the ovum (chorion), for the formation of the placenta. The portion of the allantois external to the umbilicus becomes obliterated at an early period. Of the internal remnant the inferior portion becomes developed into the urinary bladder, whilst the portion intervening between that and the umbilicus, contracts into a cord, the urachus.

Cleft member (*schistomelus*) commonly appears from between the third and fourth fingers or toes to the wrist or ankle. It is probably derived from external causes, and, as Gurlt infers from an examination of the fœtus of a dog, from adhesion to the amnion.

2. *Malformations through fusion* (*symphysis*). To these belong:

(*a.*) *Cyclopia.* In this malformation we find in the forehead a single eye, or the two eyes blended into one. It is met with under every gradation of the fusion of both eyes. The nose is either wanting, or defective, being frequently represented by an imperforate proboscis-like appendix which overhangs the one eye or the two united. The mouth is sometimes normal, sometimes misshapen—nay, the entire infra-frontal countenance may be wanting. The ethmoid, nasal, lachrymal, turbinated bones, the vomer, the superior maxillary and palatine bones, the pterygoid processes, are often all or severally absent; the anterior lobes of the brain invariably so. One explanation of this deformity is based upon Huschke's hypothesis, of both eyes being developed out of a single primitive rudiment, subsequently divided in twain by the interposition of the nasal and facial parts. An arrest in the development of these parts might then, indeed, suffice to occasion the mischief. Bischoff, however, firmly maintains that the two eyes originate at once, distinct and separate, from the anterior primitive brain-cell, and he derives the cyclopian deformity from an arrest in the development of this cell causing the too close approximation and eventual fusion of the rudiments of the two eyes. As this defective development of the brain-cell frequently causes a defective development of the anterior portion of the plastic material for the chorda dorsalis, and often for the anterior process of the first visceral arch, it would thus occasion the absence, before alluded to, of the aforesaid facial bones.

(*c.*) *Monotia, agnathus, otocephalus.* The two ears approach each other more or less below the skull, and finally coalesce. The inferior maxilla is wanting. The superior maxillary, the zygomatic, the palatine bones, along with the pterygoid processes, are either, in like manner, wanting, or else inadequately developed. The mouth is absent or very diminutive. The skull is normal, but the face small, and in brutes projects after the fashion of a proboscis. Bischoff considers this deformity referable to an arrest of development of the first visceral arch, intercepting or impairing the growth of all the said bones, and thus promoting the mutual approximation of the two ears beneath the skull. Were the internal organs of hearing implicated, the source would needs reside in a defective development of the third primitive brain-cell.

(*d.*) *Monopodia,* M. Syrenomeles,—Siren-malformation. The two lower extremities, more or less perfectly developed as to their individual parts, are blended into a single one. The pelvis, the sexual and urinary organs are wanting or imperfect; the intestinal canal is defective beyond the cæcum, and the anus invariably absent. The extremities, moreover, have revolved upon their axes, the direction of the patella and of the poples of the knee being reversed. It is founded in a faulty development of the lower end of the trunk and of its organs, the rudiments of which approximate too closely towards each other, and ultimately coalesce.

Syndactylus, aschysto-dactylus. Here the fingers or toes are imperfectly separated. It is an arrest of development, the rudiment of hand and foot, even when distinctly cognizable, not manifesting at first any division of fingers and toes.

(*e.*) *Fusion of kidneys, testicles, and ovaries.* This, according to Bischoff, is not due to arrest of development,—even these organs not originating from a single rudiment,—but rather to a defective development of the intermediate formations occasioning fusion of the rudiments.

3. *Atresiæ.*

(*a.*) *Atresia palpebrarum.* The eyelids are said to coalesce naturally towards the end of the third or the commencement of the fourth month, and to separate afterwards. Accordingly this malformation would be an arrest of devolopment.

(*b.*) *Atresia oris.* According to Burdach, the lips coalesce in the fourth month, closing the mouth until the sixth, when they again separate. According to Bischoff, however, this malformation might have a different origin. At a very early period, namely, the visceral edges of the animal layer mutually incline towards each other inferiorly, unite, and form, through the medium of Rathke's so-called inferior bond-membrane, the visceral cavity of the embryo. Not until the visceral arches break forth above, does the upper portal to the nutritive canal open, and not until still later the mouth. The atresy might, therefore, depend upon the abiding of the *bond-membrane.* In either case it would be an arrest of development.

(*c.*) *Atresia pupillæ.* Until the seventh month the pupil is closed by the membrana pupillaris, the anterior section of a vascular sac in which the lens, with its capsule, is inclosed. Its persistence determines the atresy.

(*d.*) *Atresia nasi.* According to Burdach, the nostril becomes closed during the fifth week by a saccular plug, which gradually disappears during the fifth month. Its persistence would occasion the atresy.

(*e.*) *Atresia auris externæ.* The external meatus auditorius is developed out of the posterior upper portion of the first visceral fissure. Previously to birth it is upon the whole little developed. A slight anomaly of formation may give rise to its closure, although at no period is the latter normal.

(*f.*) *Atresia ani.* The anus is not present at first, even where the terminal intestine has formed. A stand-still at this period, however, would involve the simultaneous closure of the urinary and sexual organs, seeing that their external orifices are all developed out of the primitive orifice of the terminal intestine,—the cloaca. Where, therefore, the anus is alone closed, the mischief must date from a later period, namely, after the separation adverted to has already taken place. Some physiologists believe it to be, at a certain epoch, the natural condition.

(*g.*) *Atresia vulvæ.* Probably conditional upon the turgescent edges of the external orifice of the uro-genital canal being brought into apposition, and coalescing in the female, as they ordinarily do only in the male sex, for the formation of the scrotum. Where the anus is at the same time deficient, we have here again non-development of the cloacal outlet.

(*h.*) *Atresia vaginæ,* frequently due to a preternaturally large hymen, although occasionally to a partial deficiency thereof, causing two blind sacs to overlap and compress each other.

(*i.*) *Atresia uteri,* not being derivable from the mode of development of the uterus, is to be regarded as a vice of ormation or else as the result of inflammation.

(*k.*) *Atresia urethræ,* in the male, an arrest of development, —the groove at the nether part of the penis, out of which the urethra is developed, not extending to the glans. In the fourth month the glans becomes perforated, in the natural course: if this process be checked, this part will remain imperforate.

To *acquired* anomalies of connexion belong, firstly, actual interruptions of continuity, together with their not invariable but frequent and obvious associate, *diastasis*,—that is, the parting, through loosening or lesion of continuity of the binding material of two bones immoveably connected together, and again the estrangement and deviation of the articular ends of two bones,—luxation. Secondly, agglutination and concrescence of two or more formations originally contiguous, or brought by accident or design into mutual association, and abiding contact with one another. Agglutination is effected through the binding property of recently exsuded fibrin;—concrescence through the medium of textures newly formed out of exsuded protein substances, and like unto the normal textures, for example, areolar tissue; or through the medium of such as differ in certain respects, for instance, in the degree of density of aggregation,—in the arrangement of their form-elements,—in chemical composition,—cancer, for example,—and, lastly, through the medium of vessels. This species of concrescence, in accordance with the character of the binding material, is effected by loose, filamentous adhesions, admitting of a certain degree of motion in the affected organs; or by tense and intimate conglutinations.

The adhesion of the parietes of hollow organs, and the obliteration of canals and of their mouths, represent *acquired, morbid atresy*. This originates in various ways: for example, in concentric atrophy consequent upon deficient expansive power. Thus, in ducts, it follows the extinction of the gland; in blood-vessels, the cutting off of the blood stream; or it is the effect of abiding compression and mutual contact of the parietes; or, again, it may be the result of the deposition of organic matter out of the contents of the canal, or of textural changes in its walls, produced by inflammation, cicatrisation, and the like; or, lastly, of luxuriating heterologous growths.

The coalition of the two bones in mutual contact within an articulation is specially termed articular adhesion, anchylosis.

CHAPTER VI.

ANOMALIES OF COLOUR.

ANOMALIES in the colour of organs are either essentially conjoined with or independent of change of texture. Our concern here is principally with the latter kind. We shall content ourselves with a simple allusion to the former, as the true pathological production of pigment will be separately discussed in the sequel.

The said anomalies consist in diminution, in augmented depth, or in altered quality, alienation, of colour. They affect the totality, or simply the majority of textures and organs, or, again, individual organs only, or mere circumscribed portions of these latter. Their causes, it will immediately appear, are numerous.

Diminished coloration is sometimes primitive; various organs, owing to an arrest of development, not acquiring their natural amount of colour, a condition very commonly associated with the defective development of the organs in respect both of texture and of bulk, as in the instance of muscles. We may specify a well-known species of this anomaly, namely albinoism (leukæthiopia, leukopathia), wherein the pigment is wanting in the rete mucosum of Malpighi, in the hair, in the iris, and in the choroid membrane of the eye.

It is, however, more frequent as an acquired morbid condition—in a word, as *decoloration*, *blanching*. Under this head are to be reckoned, firstly, the pallor of texture consequent upon anæmia, or upon changes suffered in various dyscrases, as chlorosis, albuminosis, dropsy, tabescent diseases, by those carriers of colouring matter, the blood-corpuscles; secondly, the blanching of textures produced by their maceration in the serum of dropsy, by atrophy, by fatty degeneration, the muscles being here the organs principally affected; lastly, that for the most part local, gradual or rapid decoloration, due, in the former case, to cessation of the development of pigment, in the latter to rapid withdrawal, or more probably to destruction, by some unknown means, of the existing pigment. As an example we may cite the topical blanching of the common integuments in coloured tribes, in parts rich in pigment (the scrotum) in whites, the progressive or sudden hoariness of the hair, &c.

Augmented or deepened coloration is, in certain coloured textures, in the animal muscles, for instance, the consequence of and the attendant upon hypertrophy. In the outer integuments, it is conditional upon the excessive development of pigment in the rete mucosum; in scars, as a purple tint, caused by the capillary vessels appearing through the texture of the cicatrix, in the embryonic stage, with its thin layer of epidermis. It is usually a consequence:

(*a.*) Of hyperæmia (congestion) and stasis, more especially where the blood is dark coloured, as in cyanosis, in asphyxia, in a typhous crasis, in an inspissated condition of the blood, consequent upon loss of serum, &c.

(*b.*) Secondly, of hemorrhage, extravasation of substantive blood into textures (apoplexy); sugillation or suffusion founded in rupture, which latter may be the consequence either of traumatic influences, such as contusion and concussion, or of excessive hyperæmia, mechanically produced; of blood stasis; of disease of blood-vessels; or else the consequence of the patency of blood-vessels, engendered by the liquefaction, the breaking down of textures.

(*c.*) Finally, of the *exsudation of blood-serum*, with an *appendage*, so to speak, of *blood-pigment*—to which category belong ecchymosis, petechiæ in decomposition of the blood, in scurvy, in putrid typhus, in acute exanthematous decomposition, in acute alcohol dyscrasis (the scurvy of drunkards), &c. As subordinate to this we may also mention those outward signs of death which assume essentially the guise of red colouring, namely, death patches, death livor, spurious sugillations. Their character varies:

1. They are dependent upon local hyperæmia and stasis, brought about during the death struggle and the period immediately following death, as the consequence of unequable palsy of the small and capillary vessels,—whence it arises that the blood accumulates in distinct patches of the capillary system various in extent, whilst in others, owing to the continued contractility

of those vessels, it is urged onward into the veins. Hence they consist in injection, and, where the usual accompaniment of the livor, from imbibition, is wanting, they are for the most part marked by being sharply bounded by a blanched texture. They are particularly frequent in the mucous membrane of the intestinal tract, and in the lungs.

2. Other death patches originate after death, being the result of cadaverous hyperæmia or hypostasis, which signifies the descent of the blood within the vessels, conformably with the laws of gravitation, to the most dependent parts. These patches are mostly of great extent, deeply saturated at their most dependent parts, and less and less so higher up. Their seat, answering to the usually supine position of the dead body, is the occiput, the posterior part of the trunk and limbs; and it includes, not alone the common integuments, but also the subcutaneous soft parts, and even the posterior portions of the viscera contained in the great cavities. Under different circumstances, they affect other regions of the body; in a lateral position of the corpse, the nether lateral half of the organs—for example, in hemi-lateral hyperæmia, the one hemisphere of the brain; in the prone position they appear in the front; in those hung by the neck, at the lower half of the subject and internally, in the organs of the pelvis and hypogastrium.

They are the more developed the greater the amount of blood and the smaller the degree of coagulation which the previous illness and the mode of death have produced in the blood during the mortal struggle. Accordingly, after acute or chronic decomposition of the fibrin in the blood, after asphyxia, they are especially marked by their rapid development after death, by their extent, and by their depth of colour.

3. A third species of death marks arises from the imbibition, by the coats of blood-vessels, and the transuding from thence into the neighbouring tissues, of blood-serum, which, owing to decomposition, has taken up a portion of the pigment of the blood-globules. In this manner are produced the livid striæ which follow the course of the subcutaneous veins in the common integuments, the red coloration of the endocardium, and of the internal membranous strata of the vascular trunks, the diffuse reddening of serous and mucous membranes, the red tinge observed in parenchymata and seemingly inherent in

their textures. Not only does imbibition pass from one organ to others contiguous,—even fluids, contained within hollow organs, as also in muco-membranous canals and in serous sacs, receive the blood-tinged serum, thereby acquiring the same cadaverous hue; or, again, the blood-tinged serum is found in the said cavities, pure, and unmingled with preexisting fluids in the form of cadaverous exsudations.

The reddening of imbibition is, of course, most readily derived from vessels, the seat of hyperæmia and of stasis; therefore very commonly from the death patches of the two species above named. The redness of injection characteristic of hyperæmia and stasis merges in, or becomes masked and disguised by, that of imbibition.

Death spots of this kind are marked by absence of injection; by the obvious cause, namely, blood being discoverable at the point of the deepest saturation, and by the stain being washed out towards the circumference.

Where the previous disease involves liquefaction of the blood plasma—they are rapidly developed, and they increase in saturation and extent in proportion as, favoured by various external influences, cadaverous decomposition gains ground.

It would appear, from the above, that death spots are, for the most part, stains resulting from a combination of hyperæmia with imbibition.

Amongst the number of imbibition stains, with the character of death marks, is to be reckoned the yellow tinge imparted to the membranes of the gall-bladder and of the adjacent membranes of the stomach and intestine by the imbibition of bile.

To *qualitative alienations* of colour belong more especially, as cadaveric stains subordinate to the above—

1. The original changes of tone in death stains to blue, purple, and violet, dependent upon the blood-crasis.

2. The brownish and greenish tints, and the dark green dye developed out of the reddening of imbibition, both in the common integuments and in other soft parts, as also in an especial manner in the intestinal membranes and their contiguous formations—namely, the peritonæum, the areolar, adipose and muscular tissues, and the liver. These varieties of colour are produced by certain gases—hydrosulphuric acid and sulphide of ammonia,—evolved in the abdominal cavity, and

within the tissues themselves. These gases react thus upon the red pigment of the blood within the tissues generally, and in the muscles most of all.

3. The dark brown, black, green, and ink-black discoloration of the spleen, from its fissured-surface to various depths, as also of the ramification of blood-vessels in the fluid sac of the stomach from the imbibition of gastric juice.

4. The more rare violet-red, iodine-coloured, diffuse lividity of the intestinal membranes.

Other preternatural colorations, for the most part equally cognizable in the living body, are, in particular—

1. The deep red tinge characteristic of thin watery blood, as also of all the tissues, down to the common integuments, in cases of poisoning with carbonic oxide and carbonic acid gas.

2. The copper-red tint of the skin in venereal stains, and in the circumference of venereal ulcers and skin eruptions.

3. The diffuse sallowness, and the circumscribed freckle-spots, termed liver-stains or ephelides, in cachexia.

4. The violet hue of typhous hyperæmia and stasis.

5. The greenish and yellowish tones of sugillation of the common integuments, arising from deep-seated extravasation of blood.

6. The yellow tinge of the solids and fluids, assuming manifold shades, the most intense of which are a brazen- and a greenish-yellow, engendered by the colouring matter of the bile, where the secretion and excretion of that fluid are intercepted, or where bile mingles with the blood, as in the typhous crasis. It is frequently superinduced by pyæmia, and occurs as the substantive and essential dyscrasy in yellow atrophy of the liver, and probably in yellow fever. As this pigment generally associates itself with the exsuding plasma, the majority of the soft parts, more especially the vascular and succulent—the secretions and incidental products of inflammation—are all dyed yellow.

7. The rust-yellow, rust-brown, black-brown, and black tints of certain organs, resulting from a corresponding granular pigment, partly contained within pigment-cells—a formation, which will, in the sequel, be considered more at large.

8. In conclusion, those anomalous dyes, produced by the assimilation of pigments, or of substances which, either with

or without the intervention of some specific influence—light, for example, enter into peculiarly tinged combinations with animal tissues. As instances of the decoloration of both fluids and solids, we may cite the yellow appearance of the urine from the ingestion of rhubarb, the reddening of the bones from feeding upon the root of rubia tinctorum, the yellowness of the skin and of the mucous membranes produced by nitric acid, the swarthy complexion which follows the internal use of nitrate of silver.

CHAPTER VII.

ANOMALIES OF CONSISTENCE.

Consistence [the normal degree of mutual cohesion and of resisting power pertaining to the elements constituting a texture] is either augmented or diminished. In either case the gradations and also the forms vary greatly, cannot be estimated but in relation to the amount of mechanical violence exerted, and exist, to a certain extent, in combination with each other.

Diminution of consistence is based upon—

1. *Loosening* of the mutual cohesion between the form-elements composing a texture, through the interposition of a fluid or solidified substance. Instances are afforded in the loosening of texture through serous effusion (dropsy), and, in hyperæmia and inflammation, through their products. Such loosening of texture is generally considerable in proportion to the rapidity with which the said products form.

2. *Atrophy,* both primary and secondary, provided the density of the texture be diminished.

3. Liquefaction and breaking down of the elementary forms of the texture, as in suppuration, in gangrene, but most especially and most variously in liquid exsudation from mucous membranes, which by its chemical properties proves destructive to the underlayer. Liquefaction of the substance of the liver, through anomalous or intercepted bile, offers another instance in point.

4. Next to these rank the softenings of certain organs, par-

ticularly of the mucous membrane lining the stomach, of the lungs, and of the brain, brought about by the resolvent agency upon the textures of a free acid. They represent those processes to which, in conjunction, perhaps, with the foregoing, the term softening ought properly to be restricted.

The abstraction of earthy salts from the bones, in rickets and osteomalacia, belongs to this class.

5. It is brought about by a transformation of textures, the nature of which is most probably a breaking up, with conversion of the chemical constituents;—for example, the breaking up of the primitive muscular fibrils, or of the texture of the annulo-fibrous tunic of the arteries, in fatty degeneration.

Diminished consistency manifests itself as irregular softening, compressibility, lacerability, maceration, liquefaction, and solution; or else as pulpiness, putrescence, friability, fragility—the latter property being frequent in the osseous system, in muscles, and in the annulo-fibrous tunic of the arteries.

Increase of consistence varies, in like manner, as to its character and cause. It is based—

1. Upon diminution of the humecting plasma, by which the texture is pervaded (water).

2. Upon hypertrophy. Those augmentations of consistence are particularly marked which depend upon true hypertrophy without increase of volume, and upon various kinds of spurious hypertrophy.

3. Upon atrophy, the reduction of volume being here accompanied by condensation,—concentrical hypertrophy,—of the brain, for instance.

4. Upon inflammation,—through the solidification and textural transformation of coagulable products;—in other words, by the issue of the inflammation in induration.

5. Upon what is termed ossification, so common in the aforesaid products of inflammation.

Increase of consistence manifests itself as preternatural toughness, hardness, rigidity. Relatively to the normal condition of the textures, it often appears less in the shape of absolute increase, than of a change in the character of the consistence. Thus the friable liver, the kidneys, under certain conditions, in spanæmia, for example, toughen through defibrination of the sanguineous fluid. The increase of consistence is often, moreover, but a seeming one, and even such only with certain restrictions. Thus the organ concerned will exercise, against ordinary external influences, a resistance exceeding the natural, and yet be powerless against more forcible impressions, because, although with increased density it has become harder and firmer, it has at the same time lost its toughness, and become morbidly fragile and brittle. Muscle affords an example.

CHAPTER VIII.

SEPARATIONS OF CONTINUITY.

They are engendered either by external and especially by mechanical influences, or else by various internal causes which may, in like manner, operate mechanically.

To the former belong:

1. Simple or complicated injuries from penetrating mechanical violence, with or without loss of substance;—incised, punctured, contused, gunshot, bitten, and lacerated wounds;—solutions of continuity occasioned by fire and cautery.

2. Imperfect and complete laceration and rupture of solid as also of hollow organs, consequent upon concussive violence,—especially when in the condition of repletion and of distension,—lesions of continuity frequently unaccompanied by perceptible injury to the common integuments and to the parietes of the implicated cavities of the body. The casting of the envelopes of particular organs caused by similar violence, as the separation of periosteum, of the dura mater, from bone, of the fibrous capsules (tunicæ albugineæ) of certain viscera, as of the spleen or the kidneys,—is of like significance.

3. Simple and complicated fractures of bones, incurvation of soft, rickety bones, casting of the epiphyses.

Separations of continuity *from internal causes are dependent upon various contingencies*. Where mechanical influence is simultaneously at work, the two influences cooperate in such wise that where the one predominates, less of the other suffices to produce the effect. Their occurrence may be rapid or slow. They are brought about—

1. By violent exercise of the voluntary and involuntary

muscles, lacerating either these or their tendons, or even affecting the bones,—in convulsions, for instance.

2. By excessive distension of hollow organs, as in laceration of the intestine, of the urinary bladder from accumulation of its contents through paralysis, through mechanical obstruction, stricture, closure, &c.

3. By hemorrhage. Here the lesio continui consists in great laceration, contusion, disruption, destruction of texture; take, for example, apoplexy of the brain, of the liver, of the muscles; the forcible separation of the strata composing a membranous organ; the loosening of the enveloping membrane of organs, of the periosteum from bone, of the tunica albuginea, through extravasated blood.

4. By atrophy. When favoured by a mechanical influence it occasions a rapid lesion of continuity in the shape of laceration,—or else, being itself caused by pressure and tension, it serves in the long run to produce lesion of continuity, more especially in the muscles and nerves. Under this head should be mentioned the spontaneous casting of normal and of morbid formations, owing to defective nutrition, as of the hair, the nails, the teeth, horny excrescences, and the like.

5. Separation of continuity is the final result of high degrees of diminution of consistency, especially in true softening, the consequence of textural disease. If, therefore, any mechanical cause be requisite at all, the slightest,—even the degree ordinarily in operation, as for example, repletion of a hollow organ, suffices to produce the effect. Amongst textural diseases, inflammation—from its effect in the loosening of tissues,—and the fatty degeneration of muscular organs, more especially of the annulo-fibrous tunic of arteries—stand pre-eminent.

6. In fine, lesions of continuity are engendered in primary textures, as well as in new growths by various processes of liquefaction and dissolution, especially by suppuration and gangrene. To this head belongs, amongst others, the spontaneous separation of dead parts, for instance of fingers, of entire limbs, of heterologous products, such as fibroid and cancerous growths. Lesions of continuity are, upon the whole, simple, or else more or less associated with loss of substance. Their cure is effected by the immediate union of the edges or surfaces of the wound, or, in the case last alluded to, by regeneration.

CHAPTER IX.

ANOMALIES OF TEXTURE.

These are the most important of all. They affect the solids and the fluids, especially the blood, in so far as certain form-elements enter as essential ingredients into its composition, and in so far as this general source of nutrition, under particular circumstances, contains and supplies formative matter anomalous in many respects as to its embryonic character and primitive forms, and also as to all its ulterior stages of development. They are commonly connected, in the relation either of cause or of effect, with various other of the anomalies of volume, of consistence, of form and of colour already discussed.

Every change of texture being founded in an anomaly of general nutrition, the proximate causes of this anomaly have to be investigated.

As such are directly demonstrable or at least deducible from analogy,—alteration of the blood, as the general fluid of nutrition, and anomalous character of the nutritive process itself. Accordingly, textural disease of the solids is in the one case the reflex of constitutional disease, in the other case a mere local ailment.

Quantitative anomalies of nutrition having been considered under the heads of hypertrophy and atrophy, the present chapter will comprise those textural diseases alone which depend upon qualitative anomalies of nutrition.

Textural diseases may be primitive arrests in the development of the texture—for example, in bones, in muscles, in pigment,—such as we may observe at any time in tissues adapted for regeneration. The majority are, however, acquired during intra-uterine, and more especially during extra-uterine life. In the former case the textural anomaly is congenital.

Textural anomalies manifest themselves—

1. As *new-growths*. The most numerous of the class.

2. As a *breaking down of texture*. The disruption involves both the primitive physiological and the new-formed pathological

textures,—the latter, by their frequent persistence, at the embryonic stage of textural development, or even in the condition of the primitive rude blastema, are particularly predisposed to this sort of conversion of their elements. To this subdivision belong, besides the reduction of textures in atrophy—besides the breaking up of textures in genuine softening, in particular the liquefaction of textures in various processes of exsudation, in suppuration, and in gangrene. And to these are yet to be added other processes which seem to be conversions of various complex substances constituting, now a rude blastema, now a definite texture,—the breaking up of fibrinous, albuminous blastemata, of muscular fibrils, of yellow artery-fibres, with conversion of their elements into fat, &c. They will be considered, together with their attendant circumstances, under the heads partly of general, partly of special anatomy.

New-growths, as already observed, furnish forth the great majority of textural affections. For, apart from their mere local characters, almost all constitutional diseases are prone to localization and to the deposition, within an area more or less defined, of products in the shape of blastemata. Even the processes of liquefaction are in part ultimately reducible to a new-growth, for example, to an exsudation destructive of the texture, to the production of acid, &c. Textural diseases assuming the shape of new-growths were formerly distinguished in a manner which here calls for a few remarks. Pathologists discriminated between:

1. Changes (metamorphoses) of textures.
2. Genuine *new* or *heterologous* growths.

With reference to this distinction which, before the adoption of the present auxiliary methods of research was highly estimated, we have to observe that, strictly speaking, the conversion of one texture into another only occurs in isolated instances, which will be hereafter specified. With these exceptions, all conversions are but seeming ones, and consist in the anomalous growth becoming developed betwixt the elementary particles and filling up the interstices of the original normal texture, so as to occasion the reduction and resorption of the latter.

This process of reduction and absorption may be so complete as ultimately to cause the original texture to be altogether replaced by the new-growth, which now presents one uniform mass, corresponding in volume with the texture expelled, or even exceeding it,—in a word, constituting a tumour.

But although this process apparently disproves any conversion, it still remains matter for inquiry whether, in a certain sense, a conversion do not take place,—whether the original but reduced texture do not, under the determining influence of the heterologous development, furnish the blastema for the new-growth? Several circumstances afford decisive evidence of a metamorphosis of this kind.

As true conversions are to be regarded—

(*a.*) The ossification of cartilages intended for permanent ones; as also of pathological cartilage—of certain enchondromata. Again

(*b.*) The fibrillation of the hyaline intercellular substance of cartilage.

(*c.*) A metamorphosis of muscular fibre into areolar fibrils, such as takes place, in the organic muscles obviously through a splitting of the muscle fibres,—in the animal muscles, probably through fibrillation of the collapsed sheaths of the primitive muscle fibres, after the breaking up and resorption of their contents (the primitive fibrils).

(*d.*) A transformation of the organic muscle-fibres into the annulo-fibrous membrane-texture of arteries.

(*e.*) A transformation of primitive muscle-fibrils, of the fibrous web upon the layers constituting the annulo-fibrous membrane-texture of arteries, into free fat (see fatty degeneration). We are not indisposed to believe in a conversion of hepatic cells into the elementary cells of medullary cancer.

Let us, after this disquisition, turn to the division of new-growths:

1. Into organized and organizable new-growths.
2. Into unorganized new-growths.

1. ORGANIZED NEW-GROWTHS.

A. OF ORGANIZED NEW-GROWTHS IN GENERAL.

These resemble normal textures, at least in their elementary composition,—and very frequently in the (secondary) arrangement of their form-elements. Where they appear amorphous, the character of the blastema attaches to such amorphous growths. They are occasionally united with unorganized new-formations, and that commonly in a consecutive manner, the latter supervening upon the new-growth, as in the instance of so-termed ossification in the shape of concretion or incrustation.

New-growths present great and important differences in relation not alone to the form-elements—especially cell and fibre,—but also to the secondary arrangement of these elements into a texture.

Nor do they differ less widely and essentially as to origin and development. In this respect they often, it is true, follow the laws of cell-formation—cytoblastema, elementary granule, nucleus, cell, fibre. The field, however, is equally extensive of fibrillation out of nuclei and granules, and especially that of the independent development of fibre directly out of solid blastema, intercellular substance, primitive structureless membrane, and membranaceous coagula. Compare, with reference to this and to what next follows, *Blastemata.*

With reference to the grade of development attained by their elements, new-growths are classed, if such a classification be feasible, considering the variety of elementary bases coexisting in a single growth, and the want of uniformity in their ulterior development, as follows:

1. Such as exist in the condition of formless liquid, or at that phase of coagulation—the consolidating blastema. They are susceptible of further development, or they abide at this stage, many ultimately breaking up. They comprise some very malignant new-growths—for example, tubercle.

2. Such as attain to nucleus and cell-formation only,—perhaps to fibre- or caudate cells. They consist of isolated cells within a fluid, semi-fluid, intercellular substance (pus; colloid, encephaloid substance); or, again, the cells are imbedded in a paucity of firmer, amorphous intercellular substance, which acts as a bond-mass. Along with them are numerous nuclei and elementary granules—embryonic formations readily broken up. Some of the most malignant new-growths are thus constituted.

3. Such as have their texture represented by fibres of different kinds, variously arranged, and arising out of cells, nuclei, elementary granules, or directly out of blastema. To this class belong many quite benign, and a few eminently malignant formations: for example, fibrous cancer.

4. New growths, which, in their full development, consist of fibres, cells, nuclei, blastema, although the disposition and the mode of development of these elements may greatly vary. They include new formations, both benign and the reverse. The progressive development of the form-elements is accompanied by a succession of chemical changes. Under every mode of development the reactions vary with every phase, from the primitive blastema to the completion of a texture, the difference between the perfect texture and the primitive blastema being very marked indeed.

With reference to the development of blood-vessels, new growths are either *vascular* or *non-vascular*. The former present every gradation, from poverty in blood-vessels to luxuriant vascularity. Nor does the number of its blood-vessels stand in any direct relation either to the bulk or volume of the new growth, or to the stage of its development in other respects. Accordingly, there are, on the one side, new formations of very considerable magnitude, which, devoid of all blood-vessels, vegetate freely in the cavities of the body—for example, the frequent fibroid concretions in serous cavities, certain cancers, &c. On the other side, there are blastems in which blood and blood-vessel-formation so predominate that the new growth consists of little else. (See *Pathological Blood-vessel-Formation.*)

With reference to their state of aggregation, new formations are either *fluid* or *semifluid,*—for example, the plasma containing dropsical fluids, pus, ichor, encephaloid fluid, colloid, the gelatinous substance of collonema, of areolar cancer, &c., or they are solid.

Not long since there existed a classification of new growths, which, though not altogether available, is nevertheless deserving of mention—namely, their division into *homœoplasiæ* and *heteroplasiæ*. The former (according to the earlier views of Meckel) are repetitions or imitations of normal textures, the latter alien to the normal composition of the organs and textures. With respect to this division, it is to be observed, that:

(*a.*) The chief argument against the assumption of heteroplasiæ is afforded in the evidence recently obtained, that all new growths essentially imitate normal formations, not alone in their origin, development, and growth, but also in their chemical composition.

(*b.*) Even the secondary arrangement of their textural elements, that is, their coarser texture, very frequently offers analogies with normal textures. Finally, their general aspect exhibits to the naked eye analogies which formerly served for the basis of certain classifications and denominations. Take for example the comparisons with various glandular structures made by Abernethy and others.

(*c.*) It might appear from the above, that homœoplasiæ alone existed. Still, in many heterologous formations the external aspect, the structure, and even the textural elements, especially cell and fibre, differ, not only in themselves, but in the progress of their development and in their chemical composition, so materially from the normal type, that the existence of heteroplasiæ cannot be altogether rejected. As regards the relation of new growths to normal texture, it should be stated that,

1. The heterologous formation lodges more or less uniformly between the elementary parts of a texture, the latter becoming infiltrated. The mass (and commonly the volume also) of the organ increases—*false hypertrophy.*

2. Where, on the other hand, the heterologous formation is developed and increases from an interstitial point, or even from an originally circumscribed infiltration, so that at its circumference it rather displaces than involves or embraces the elements of the affected organ, and so that its periphery becomes more or less sharply defined, it forms an individual independent heterologous mass, termed a tumour.

The distinctive characters of the two are, however, by no means strongly marked. Akin to the above division is another, almost essential to the medical practitioner, however little tenable in a scientific point of view, namely, into benign and malignant new growths (benign and malignant tumours). The connexion between the two classifications consists in this, that, with certain exceptions, homœoplasiæ appear to and actually do answer to the character of benign, whilst a grade of malignancy may be predicated of a new growth proportionate to the degree of its heterogeneous nature. In this classification it is essential to determine,

1. What constitutes a benign, what a malignant new growth?

2. What are the distinctive marks of the one and the other?

In the first place, we would signify by malignant new growths, those the origin and continuance of which either are essentially bound up with, or else eventually lead to, a definite constitutional dyscrasis, a general disease giving rise to a peculiar impairment of nutrition, and a multiplication of specific new growths. No new formation is, herefore, in itself malignant, but becomes so either through a specific, pre-existent, and predetermining, or through a consecutive, general dyscrasial affection. This is perhaps the proper explanation of a malignant new growth. It does not preclude that occasional purely local relation of a malignant formation upon which the cure of the latter, spontaneous or artificial, often depends. It will be seen that whatever else is adduced as an attribute of malignant tumours ceases to be distinctive.

(*a.*) It is very difficult to recognise a constitutional disease as a definite one reflected in a new-growth, and to discriminate between this and a cachexia engendered by the luxuriation and ichorous vent of a new growth essentially local, and pronounced benign. Besides, the constitutional affection may as yet be altogether wanting. Even where several growths coexist of the same character, or rapidly succeed each other, they need not necessarily be based upon any general dyscrasis. They may be simply so many mere local occurrences.

Certain other characteristics are indispensable for a diagnosis, and, at the same time, difficult to establish: for example, that—

(*b.*) Benign growths are curable by extirpation, whilst the malignant recur at the same spot, or at other spots, or even at both.

In opposition to this, it is to be urged, that many benignant new growths recur after extirpation, where the disposition to them remains, whilst, under certain conditions, many a malignant new growth does not recur, but enters upon a spontaneous process of retrogression, and becomes extinct.

(*c.*) Malignant new growths have a marked tendency to draw within their formative range—to convert to their own similitude—contiguous and neighbouring textures.

It is to be observed, on the other hand, first, that the most malignant growths thrive and flourish as independent tumours upon a new-formed vascular apparatus of their own, without otherwise molesting the surrounding textures than by forcing them from their positions; secondly, that where the original normal textures merge in the heterologous growth, this is brought about in the malignant, precisely as it is in the benignant ones—namely, by a conversion in the sense before adverted to; that is, through reduction, disintegration, and resorption of the normal textural elements.

(*d.*) When malignant growths have attained their highest point of development, they break up and enter upon a process of softening, which implicates or involves surrounding and included textures, and thus serves to exhaust the organism.

In connection with this process, the following subjects for consideration suggest themselves: namely,

1st. This so called stage of metamorphosis—this breaking-up very frequently fails to occur, even in the most malignant new growths.

2d. That apart from the general difficulty of establishing the epoch of the highest development of a new formation, the act of breaking up should seem a fortunate event as regards the growth itself, which, by virtue of the elementary transformations thereby engendered, becomes deprived of its importance, and in many instances is excreted from the body.

3d. That this metamorphosis for the most part simply implies inflammation terminating in ichorous degeneration, and death of the new growth.

4th. That this destructive process frequently attacks the surrounding textures merely in the character of suppuration, and that,as such, it may, whether based upon a benignant or upon a malignant new formation, either exhaust the organism, or, on the contrary, lead to a cure,—to the expulsion of the heterologous product.

5th. That where a fresh development of heterologous substance is excited and kept up in neighbouring parts by inflammation connected with sustained ichorous secretion, the malignancy of the growth may be at least strongly suspected.

(*e.*) Malignant new-growths are said to abound in albümen and casein; benignant new-growths, in fibrin and gluten.

Were such a distinction of new formations not rendered nugatory by the convertibility of those organic substances, it would become in a great measure deprived of its value by the number and weight of the exceptions,—for example, those of fibrinous tubercle, of fibrous cancer, the composition of which is marked by a considerable amount of gluten, &c.

(*f.*) Homœoplastic formations are, for the most part, benignant, heteroplastic growths malignant.

Supposing such a distinction admitted, its utility would still be doubtful, seeing that in a given case the decision frequently depends upon the method followed in the examination, and upon individual opinion; and again, that in many new formations, homœoplasia and heteroplasia coexist in various gradations. For the more marked repetitions of normal textures, namely, areolar, cartilaginous, osseous new growths, the character of benignancy might indeed be predicated; whilst, on the other hand, certain forms of *malignant* fibrous cancer bear so close a resemblance to the *benignant* fibroid new-growth as to set discrimination at nought.

Microscopic analysis, therefore, from which important disclosures in relation to the diagnosis of benignant and of malignant growths, and tenable grounds for the establishment of a system were expected, has in reality thrown but an uncertain light upon the subject.

Certain new growths are especially intended for the more or less perfect restitution of loss of substance, howsoever occasioned. These regenerated textures are sometimes perfectly identical with the lost ones, in formal and chemical composition, as also in function; sometimes entirely dissimilar. The latter kind are represented in scar-texture, which, again, may have an evanescent existence, as in *provisional cicatrix*, out of which is developed, and which merges in, a texture identical with the lost one; as, for instance, the fibroid scar-texture that ensues upon loss of substance in the bones of the skull; the scar-callus occurring at the point of a lesion of continuity in a nerve. Or again, the cicatrix may be permanent, consisting throughout of a fibrous texture of various degrees of perfection, in which the elements of the lost texture are never reproduced: such is the muscular, the glandular cicatrix.

New growths once established either sustain themselves without alteration of bulk, or else wane and shrivel in various ways, or even disappear altogether. Products of inflammation, even such as have assumed a textural character, unquestionably become reabsorbed; so, in like manner, do new growths of embryonic structure.

Again, they liquefy under various transformations of their chemical components, or they become diseased in manifold ways.

Finally, new growths increase. This increment takes place through juxtaposition; that is, through the accession of blastema upon the periphery of the existing structure. Such is the growth of non-vascular formations, especially of those which do not rise above the lowest grade of development; as, for example, tubercle. Or the increase takes place through the intussusception of new blastema from those blood-vessels of the diseased organ which supply the new growth, or from an adventitious vascular apparatus newly developed for the supply of the new formation. Finally, an increase of volume may be based upon the variety of chemical conversions attending the development of textural elements out of blastema, and, in vascularized heterologous products, attending the growth of those elements themselves.

Growth and intrinsic development by no means keep pace with, but rather stand in an inverse ratio to, each other. Rapidly vegetating heterologous growths are mostly distinguished by an embryonic structure.

Upon the rapidity of its growth depends, in a great measure, the degree of influence exercised by the heterologous product upon the affected organ, upon its vicinity, and upon the organism generally.

This influence, considered locally, consists in pressure and tension of textures and of entire organs; in displacement and extinction of textures; consequently, in the production of pain, and in embarrassment or complete hindrance of function.

The influence upon the entire organism is sometimes deducible from that which is local, shaping itself differently according to the different seat of the new formation. In the instance of heterologous products luxuriating by growth and multiplication, this influence consists in causing the wasting of organic matter and of power, or in the establishment of a consecutive dyscrasial state. This latter may be brought about in a twofold manner:

(*a.*) Either through the withdrawal from the fluid of nutrition of some particular substance employed as a material in the heterologous structure—as in defibrination of the blood and œdema in tubercle—in dropsy consequent upon albuminuria.

(*b.*) Or else in a positive manner, namely through reception into the blood and lymph of substances generated in the interchange of matter that constitutes the nutritive process of the heterologous product, and still more through reception of the heterologous matter itself, in the shape of intercellular substance, or of elementary cells, and the like. This directly leads to contamination of the fluid of nutrition, and thereby to a dyscrasy reflecting the character of the heterologous growth. It is the more speedily brought about where circumstances are generally favorable to endosmosis or resorption, and particularly so in the locality of the heterologous growth, where this latter is bulky or highly vascular, or situate in organs rich in blood and lymph-vessels, where its mass (its intercellular substance) is more or less fluid. It occurs, however, in heterologous growths, both solid and poor in blood-vessels, when their texture has become disintegrated and liquefied by hyperæmia and inflammation. To sum up, new growths possess sometimes a general, sometimes a local, character. Nay, one and the same new formation may, at various, successive periods, acquire now the one, now the other character. A growth, originally of general import, may in particular assume a local one instead.

New growths vary considerably as to the organs and texture which they affect by preference; each possessing, in this respect, a scale of frequency of its own. Some organs are preeminently subject to one particular kind of new formation.

Certain new formations become developed and subsist unmistakeably in concurrence; certain others never cohabit, the presence of the one serving to exclude the other,—the appearance of the one arresting the development of the other. Exclusiveness or repulsiveness of this kind is, as might be expected, mutually evinced by new growths based upon dyscrases of opposite characters. On the other hand, new formations rooted in kindred dyscrases, do exist confederately, and purely local new-formations enter into every phase of combination.

Let us now turn from the consideration of confirmed new formations to that of their blastema and of its metamorphoses.

OF BLASTEMA AND ITS METAMORPHOSES, WITH AN ESPECIAL REFERENCE TO FIBRIN.

The blastema for pathological new growths ultimately proceeds from the general fluid of nutrition, the plasma of the blood. Accordingly, its source is that out of which all normal textures are developed. Its bodily detection and demonstration in its simple, primitive form, are, however, mostly a matter of difficulty, except, perhaps, in cases where it is somewhat copiously produced, in the train of peculiar and often rapidly fatal processes, which may be experimentally analysed grade for grade,—for example, in inflammation and hyperæmia. It exudes through the parietes of vessels wherever capillaries exist, or it appears as an endogenous segregation from the blood within the circulating system. In rarer instances, it is deposited by extravasation out of lacerated vessels.

The blastema is originally fluid, and it may either abide in this condition or solidify. The earlier or later solidification, that is, its becoming a fixed elementary body, and the degree of the resulting density and consistence depend mainly upon the presence of coagulable protein, and upon the degree of its coagulability, as also upon the absence of those counter-checks to coagulation, alkalies, acids, and certain salts.

Rapidly solidifying blastemata, especially when products of inflammation, are very commonly termed plastic,—improperly, however, because coagulability of the blastema stands by no means in any direct relation to the faculty of development. Many blastemata, distinguished for their coagulability, do not rise above the lowest grade of form-development, and not alone do they stop at the grade marked out by the process of coagulation, but their ulterior tendency is to liquefy. An example is afforded in tubercle.

As a fluid, primitive blastema recently secreted is amorphous.

Sooner or later, however, it is marked by the development of form-elements, in the shape of molecular granule, nucleus, cell. Solidified blastema is either at the outset amorphous, or displays, from the moment of coagulation, certain, and, indeed, higher elementary forms,—more especially fibrillation.

The blastemata are colourless, or they assume the tint of the plasma, or they are of a reddish gray,—the fibrinous, of various tones of yellow,—the albuminous, whitish, particularly when fat enters simultaneously into their composition,—or they display various shades of red from adhering blood pigment, or from the presence of blood-globules, &c.

Chemically considered, all blastemata for pathological new-growths are protein-compounds, for the most part in various degrees of oxydation.

The main conversion which the blastema undergoes is its development into textures. It is capable, however, of abiding in its rude primitive condition—of remaining dormant—or of breaking up, or, lastly, even of becoming reabsorbed.

Before we proceed to consider these several attributes of blastemata, it seems desirable for us to render ourselves familiar with the main conditions for its development or non-development.

If, participating in the current opinions as to the conditions necessary for the development of blastema, we admit

(*a.*) A faculty of development originally and essentially inherent in the blastema, and inseparable from the idea conveyed by the term.

(*b.*) The necessity of certain outward and general conditions, particularly a mean temperature, the presence of water (moisture) and of oxygen.

(*c.*) *The necessity of extant life in the textures* into which the blastema is effused, and *à fortiori*, in the individual. In necrosed textures no development takes place at all.

(*d.*) The necessity, in order to become developed, that the blastema should abide in close contact with the living textures; for beyond this the influence of the vital power certainly appears to be limited. The development of blastema usually commences close to the living textures, and bulky effusions of blastema remain in a backward state when removed from these textures, lingering either in their rude primitive condition, or at the stage of form-development, determined by coagulation, or, lastly, breaking up.

(*e.*) The specific influence exerted by circumjacent textures upon the mode of development, and upon the form of blastema. We know that, in the act of nutrition, of regeneration, even in pathological processes, blastema in areolar tissue becomes developed into areolar tissue; blastema in serous membranes into areolar tissue, nay, even into serous layers and sacs; blastema in bone, into bone; we know that, in tumours, fibroid textures often imitate the texture of the organ; that fibroid tumours of the uterus, for example, represent the elementary forms of organic muscular fibre;—that in bones, cartilaginous new growths are wont to assume the form of enchondroma.

All this generally admitted, the failure of such influence does not, as regards many, and these the more momentous cases, appear satisfactorily to explain either the non-development of blastema, its tarrying in its rude primitive condition, its arrest at an inferior stage of embryonic development, its disintegration, or its development to unwonted heterogeneous textures. This becomes the more obvious if, in relation to the aforesaid conditions, we reflect:

(*a.*) That, as a rule, the absence of moisture is not absolute, and that it is also in other ways conditional.

(*b.*) That the absence of an adequate, general, and specific influence in the circumjacent textures can never be assumed directly, but only through the one-sided conclusion that, notwithstanding the existence of other requirements, a blastema has failed to become developed, a fact which might admit of a very different interpretation.

Thus, to discuss a matter of the greatest importance in the arena of facts, the sojourn of certain fixed blastemata,—for example, tubercle—in the primitive condition, is not ultimately referable to the absence of moisture; the absence or rather paucity of water depending upon the high degree of coagulability proper to the said blastema. This coagulability must, however, be inherent in the blastema itself. Again, there are blastemata which never get beyond the embryonic grades of development, —for example, the pus-blastema, the blastema of medullary and of calloid cancer. Deficiency of vital power, or of determining influence on the part of surrounding textures cannot, in every instance, furnish grounds for the non-development of blastema. Thus we see very minute portions of blastema,—for instance, of tubercle—in robust individuals, in the closest contact with, nay, in the centre of vigorous textures, undeveloped. On the other side, in a very low degree of vital power, where one might rather expect little or no blastema to be produced, we meet with enormous masses of it under various forms of heterologous growths, engaged in the process of development.—The phenomenon so commonly regarded as an arrest of textural development,—founded in deficiency of vital energy, of adequate working power,—namely, fibrous callus in the regeneration of bone, —cicatrix in muscle, &c.—is, we think, interpretable as qualitative alienation, the blastemata not abiding at the embryonic stages of development of the textures to be regenerated, but forming into other textures, perfect after their kind.

Still less is this *deficiency* calculated to illustrate that qualitative variety in the development of blastemata, exemplified in so many heterologous growths. How should we therefrom apprehend the derivation of a cyst, of an areolar-carcinoma and the like. How often should we not be driven, instead of *deficiency*, to assume an equally unintelligible *excess* of power, where we find, in textures of an inferior grade, new growths developed, the elements of which belong to textures of a higher order.

These remarks of themselves lead to the conclusion:

1. That the abnormal development of the blastemata is founded, not in a deficiency, but in an anomaly, of determining influence.

2. That the different blastemata themselves, at the outset, possess indwelling properties of their own. We can have little hesitation in establishing, as a basis of the doctrine of new formations, a native anomaly in the blastemata, this being practically demonstrable. Such, for example, are the various morbid relations of protein substances, and in particular the anatomically demonstrable anomalies in the constitution of fibrin in the blood itself, with which anomalies the different exsudations (as blastemata) correspond both as to form and chemical composition.

In this manner certain blastemata bear, in their primitive character and composition, the grounds for their non-development,—the seeds of their dissolution,—for example, croupous fibrin,—tubercle,—pus-blastema.

Other blastemata, on the contrary, possess the in-dwelling faculty of development in so exalted and inextinguishable a degree, as to form, in large serous cavities, into free aggregations of blastema without any abiding contact with textures—free fibroid concretions.

Areolar new growths are so frequent, simply, in our opinion, because their blastemata are so frequent, and their production consequent upon so many different processes of exsudation.

The blastema for animal muscular fibre appears, on the contrary, to exsude only in the normal process of nutrition, or where this process is exaggerated to hypertrophy.

It is very common for mixed blastemata to exsude. Hence the frequent coincidence, in one and the same new formation, of such various elementary forms, and of such different modes of development.

Primitive anomalies of blastema may be occasioned in a twofold manner:

(*a*.) They may be rooted in a general dyscrasy of the sanguineous mass. The effusion of blastema coincides with manifest anomaly of general nutrition. The blastema bears the impress of dyscrasial adulteration. This is particularly the case with blastemata deposited in mass as inflammatory products. Indeed, the copious production of blastema in reduced, enfeebled subjects, admits of no other conclusion than that of a dyscrasial condition as the source of such effusion; the specific character of the latter being simultaneously manifested by its obvious preference for particular organs.

(*b*.) Again, the said primitive anomalies may, where the general crasis is untainted, be based in an altered admixture of the plasma (the blood) owing to local changes of innervation; or else in an anomalous act of nutrition, for example, inflammation.

In the former case, the blastema is a symptomatic manifestation of a constitutional disease, and is of general—in the latter case it is of mere local—import. The same blastema, pus, cancer blastema, for instance, may, in one case, imply general, in another, local disease.

A new growth would, however, be equally of local significance if it resulted from a blastema of originally normal character through an anomalous determining influence on the part of the nerves or textures, or of an anomalous interchange of matter (resorption, &c).

Amongst the many causal relations, the mode of operation of which is unknown, mechanical influences are by no means the least frequent originators of anomalous blastemata through local changes of innervation and of textural influence.

We shall now leave this discussion, and proceed to an inquiry concerning the *metamorphoses* of blastema. Amongst these, the first rank is taken up with the development of blastema into textures. Here *solid* blastemata, as the groundwork of pathological new growths, present so great a difference from *fluid*, that the two must, as far as possible, be separately considered.

The latter are all developed according to the laws of the cell-theory, whilst the former disclose, besides a variety of granule and of fibro-nucleus formations, a direct, and, for the most part, preter-physiological development, in various ways, to higher elements in the shape of fibre.

Blastemata of this description are represented by coagulated fibrin and coagulated albumen. In the identity, however, of the process of development in both, fibrin is preeminently adapted for experimental study, owing to the greater frequency of its occurrence, especially in an aggregate and bulky form. Under certain conditions, its coagulation alone suffices to constitute textural formation. We shall, therefore, do well to preface an enquiry into the nature of solid blastemata by the study of coagulate fibrin in its relation to the doctrine of exudation and of blood disease.

COAGULATED FIBRIN.

The simple coagulum met with in the heart or great vessels after death, and in blood drawn from blood-vessels during life, furnishes the chief groundwork for this inquiry.

These coagulations, which vary essentially, both as to external appearance and to elementary constitution, form the basis of the different qualitative fibrin crases. We should, however, begin by stating that the individual forms seldom, if ever, occur in their perfect simplicity, owing both to the mutable nature of the substance, and to the fibrin not becoming throughout equably influenced by the disease. This corresponds to the numerous exsudates composed of differently constituted fibrinous materials, as also to the frequent impurity of blastemata in general.

The more important forms, described from the most perfect specimens, are as follows:—

1. Fibrin taken from the dead bodies of healthy individuals, presents tolerably compact and tough, moderately adhesive coagulations of a yellowish white. These are separable into membranous layers, and their torn surface exhibits a delicately villous character. Viewed under the microscope, they display a transparent basement, capable of membranous expansion, or else stratified. Upon this is a dense felt, freely erect at the edges of the preparation, and consisting of very minute, very elastic, ramified fibres, visible in black outline, and rapidly soluble in acetic acid. Hard by on the preparation, are seen numerous round polished nuclei, which, when treated with acetic acid, are brought more distinctly into relief. Beside these lie scattered minutely granular, dull, round, and elliptic nuclei, and similar cells, the size of pus-cells, colourless blood-globules, lymph-globules (fibrin-globules, according to *Mandel*), the same form-elements which, in exsudates, are termed plastic corpuscles (Bennett) exsudation cells (Henle). (See also Pappenheim, Addison, and others).

The soft, jelly-like coagula of so-called *spurious fibrin*, found to accompany the above-mentioned compact coagula, and in certain cases to constitute the whole of the impoverished fund of fibrin, show the same composition. They constitute, we think, a transition form from albumen to fibrin, of great moment in relation to the normal process of nutrition.

2. Fibrin the coagula of which, though of the ordinary appearance, possess the property of adhesiveness in a more marked degree, and frequently inclose not inconsiderable quantities of serum. Examined with the microscope, they present a laminated basement, and one splitting into fibres, flattened or roundish, rough, and firm, or resembling organic muscular fibres; or else a membranous basement invested with delicate wavy fibres, upon which, amongst elementary granules, are seen

numerous round, black-edged nuclei, sometimes rod-shaped, or drawn out into fibres, and again, more especially in the moisture poured out, dull, round, and oval nuclei, and analogous cells. This fibrin enters—along with rudiments of the preceding one—into frequent combinations with those about to follow.

This and the preceding fibroid together furnish the basis of numerous areolar or fibrinous new-growths, whether simple or combined with other blastemata, both within and without the vascular system; textural development having set in with the process of coagulation itself. The exsudation of the last specified form of fibrin is especially wont to accompany morbid processes; for example, inflammation, and frequently in considerable abundance. It might be designated, for distinction's sake, as *plastic*, or *organizable fibrin.*

3. Fibrin, the coagula of which are marked by opacity, and by a dull-white aspect shaded with yellowish, or with yellowish-green. They frequently include, besides blood-serum, blood-corpuscles in considerable amount, thus giving proof both of augmented coagulability, and greater rapidity of coagulation. They are opaque, and of various shades of red. Microscopically examined, the coagulum presents a stratiform or fibro-laminated basement, or else a faintly striated membrane, both being, however, opaque, owing to delicate granulation (Punktmasse). Upon this, as also in the serum, are seen a vast number of nucleus-like formations, of developed, dull granulated nuclei, and of similar more or less developed cells. Frequently the coagulum appears to consist altogether of the two last-mentioned elements, with a proportion of granulated structure. The nucleus-formations all manifest the usual neutral relations towards acetic acid. This fibrin possesses little adhesive property.

4. *Fibrin* presenting in a higher degree the morbid characters manifested in the preceding variety. The coagula are extremely opaque, and, where they inclose no blood corpuscles, of a more marked greenish-yellow tinge. Frequently, however, they do inclose vast quantities of blood corpuscles, and are of a reddish-gray or a reddish-brown, denoting rapid coagulation. Upon a closer examination, they are found to consist of a compact, delicately granulated mass of nucleus and cell formations

(assimilating in various degrees to the pus-cell and pus-nucleus), all held together by a tough amorphous intercellular substance. There is neither fibrous tissue nor any fibrillation. These coagula possess still less of a cementing property.

These two latter forms we would designate as *croupous fibrin.* Here the fibrin borders upon that in pyæmia, and has the croupous character. The cells and nuclei included in the coagulum are genuine pus-nuclei and pus-cells. Other morbid conditions of fibrin—for example, the milky white opaque fibrin are of little moment as regards the present subject. They will be considered under the head of Crases.

These forms of fibrin possess, from the very first, an indwelling proneness to textural formation, and a disposition to molecular disintegration—nay, they have already entered upon both the one and the other transformation. The fibrin 1 and 2, are organizable; the fibrin 3 and 4 suffer disintegration; portions of the fibrin 1 and 2 that mingle with it being alone susceptible of textural transformation, as is so frequently witnessed, extraneously to the vascular system, in exsudations of a kindred stamp. Fibrin 4 presents no definite coagulum at all.

These forms of fibrin correspond in some measure with Mulder's gradations of the oxydation of protein. Here, however, chemical analysis has assuredly not kept pace with anatomical facts.

Coagula assuming as it were the form of intercellular substance, are liable to both kinds of metamorphosis. The differently apportioned nucleus and cell-formations here play a subordinate part, their importance varying, as has been stated, from the nucleus employed in the fabric of textures, to the true pus nucleus and pus cell. Hence they are the manifestation either of a quantitative endogenous development of textural rudiments, or else of a qualitative affection of the plasma.

1. The structural transformation comprised in the process of coagulation, consists in the afore-mentioned diverse fibre- and membrane-formation. The nuclei themselves, sometimes appear elongated into rod- or perhaps spindle-shaped fibre-stems. In the cells the caudate form of development is seldom observable.

2. The second metamorphosis is disintegration. It is foreshadowed in the granular mass that enters into the coagulation.

After probably a brief interval, the entire coagulum resolves itself into a pulpy, cream-like whitish, or yellowish white, or, if containing blood corpuscles, into a proportionally faint reddish gray, reddish brown, or chocolate coloured liquor, pregnant with granulated substance along with the nucleus and cell formations originally admitted into the coagulation, and becoming, where the latter are numerous, relatively analogous to, and where they assume the character of pus nuclei and pus-cells, identical with, pus. This breaking down may, under certain external conditions, unfavorable to textural formation, or owing to some indwelling peculiarity, affect fibrine generally. In croupous fibrine it is of unfailing occurrence. The results of Gulliver's experiments concerning the liquefaction of fibrinous coagula, under the sustained influence of the animal degree of heat, out of the animal body, are not applicable to the process as occurring within the living body, where certain kinds of fibrin of necessity become converted into textures, whilst others as invariably liquefy.

This process is witnessed with especial frequency in the coagula occurring within the heart, and which Laennec designated as "végétations globuleuses," as also in the coagula occurring within blood-vessels, both great and small.

Liquefied fibrin is capable of undergoing inspissation and cretaceous conversion.

Other transformations of fibrin are:

3. The abiding of the organizable fibrin at the primitive stage of formation, and its eventual extinction. Here the coagulum is, with loss of its moisture, reduced to a compact, unyielding, semi-translucent, or opaque and horny substance. It is capable of eventually ossifying.

4. *Fatty conversion*, in the shape of a reduction to fat-molecules of various circumference, a metamorphosis which coagulate fibrin shares with liquid and coagulate albumen.

5. Within the vascular apparatus solidified albumen, of whatever form, may again become incorporated with the circulating fluid. Where this liquefaction of the coagulum is not the consequence of inherent disposition, it is wrought by gradual solution in the plasma, becoming, so to say, corroded, layer for layer, by the liquor sanguinis. Examples offer in the progressive resolution of solidified vegetations upon the heart's valves,

or of the thrombus in arteries. It corresponds to the resorption of the consolidated fibrin of exsudation and of extravasation.

We have hitherto expressly restricted ourselves to an enquiry concerning the consolidation and the metamorphoses of fibrin *within the vascular apparatus*, as exemplified in the diverse spontaneous coagulations which occur in the heart, not rarely during life; and again in coagulations within the larger vessels (more especially the veins), and also in the capillaries.

The relations of the fibrin of exsudation are precisely the same.

The organizable nature of the fibrin of exsudation might be confidently assumed a priori; it is, however, as shown under the head of hemorrhage, directly demonstrable by facts.

Contrasting the frequency with which solid blastemata constitute the basis of pathological new-growths, with their rareness in the physiological condition; reflecting, at the same time, upon the predominance of cell-development in physiological structures; and lastly, upon the absence of fibrin in the embryo, we feel somewhat disposed to concur with Zimmermann, in regarding fibrin as a genuine excretive formation; a substance carried by oxydation to the verge of disintegration,—albumen worn out by oxydation, and associated with albumen for the purposes of nutrition, only in the shape of pseudo-fibrin.

METAMORPHOSIS OF BLASTEMA.

1. Textural development.—Organization.

Solidified Blastemata, at their very development, either constitute various pure and unmingled new growths, or enter in the shape of intercellular substance, basement- and bond-mass, as the stroma into the composition of complex heterologous structures. Their development is, for the most part, foreshadowed in the types cast in the process of coagulation, and which were partly discussed in the foregoing chapter.

The principal abiding form-element that enters into the composition of new growths is the *anastomosing, delicate fibrous network* of consolidated fibrin. This, together with a hyaline intercellular substance, speckled throughout with shining nuclei, we have seen in old inflammatory indurations in the brain, as also composing an extensive fibrous cancer in the stomach.

Solid blastema either appears originally as a compact mass, or else takes up a considerable amount of moisture, and establishes a sort of skeleton-work with variously shaped gaps, offering a specific type of much interest. From a central mass, namely, arises a trelliswork, the rods of which are sometimes isolated, sometimes anastomose with each other, constituting a network with largish, and for the most part, oval meshes. This type characterises in particular the opaque accumulations found upon the internal coat of arteries, as also certain fibrous tumours, especially when seated upon the dura mater. Or, again, solid blastema assumes the form of a *membrane,* either superficially spread out, or folded and rolled up in a tubular form,—a *cylindrical* fibre.

The blastema is here amorphous, laminated; or it presents upon laceration, a striated, fibrous aspect. It may assume, owing to the presence of elementary molecules, various degrees of opacity, or a granulated look; or it may display crystalline clearness. Lastly, it may or may not include nuclei and cells, in various proportions.

Solid blastema of each of the specified forms is worked out into fibres by splitting:

1. Either *directly into areolar fibre and fibril,* or else

2. Into flat, ribband-like, rough surfaced, jagged, or into roundish, oval, mostly felt-like fibres, of from $\frac{1}{100}$ to $\frac{1}{85}$th of a millemeter in their broad diameter.

3. *Into fibres perfectly identical with those of the organic muscles.*

By renewed splitting, for the most part commencing at their ends, the two latter kinds may give rise indirectly to *areolar fibrillation.*

Where there are nuclei present, engaged in the development into oblong nuclei, the splitting takes place in the direction of their longitudinal axis.

The aforesaid *cylindrical fibres* represent little tubular bodies of from $\frac{1}{100}$th to $\frac{1}{10}$th of a millemeter in diameter, which end either in bulb-like dilatations, or in sharp points, frequently inosculate, and constitute a wide-meshed villous network. Their parietes are formed by a transparent, structureless, often wavy membrane, in a single or double fold. Their contents are elementary granules,—in hemorrhagic blastema, pigment granules additionally,—nucleus formations, cells, together with an amphorous blastema in varying quantity. Cylindrical fibre at its parietes becomes areolar fibril, or perhaps this fibrillation is first developed in the blastema within the canal, as a delicately fibrous wave-curled axis-cylinder. Such fibres are commonly coincident with the primitive forms of blastema, more especially with the trelliswork described. We have frequently examined them, and we regard them as analogous with the cylindrical formations occurring in fluid blastemata (Engel's germ-tubes.)

Other kinds of fibre arise directly out of splitting, but more slowly, it would appear, and only after the blastema has entered upon essential chemical changes (as partial glutinous or horny conversion.) They are characterised by their neutral relation to acetic acid, or at least by their stubborn resistance to its influence. Through progressive transitions they ultimately attain to uniformity with elastic and nucleus fibre with which they further accord in blackness of outline, in solidity, and in elasticity. Of this nature are

1. *A transparent fibrillation for the most part solid,* the fibre varying in diameter from that of the areolar fibril to one of undefinable minuteness.

2. A fibrillation in black outline, vibrating *in lengthy deviations.*

3. *A twig-like fibrillation arising out of a short stem,* with black contours.

4. *A fibre-felt,* resembling the intercellular substance of reticulated cartilage.

The appearance in the blastema of *roundish gaps,* created by resorption, is likewise deserving of notice. In this manner solid masses of blastema acquire a *porous, honeycombed aspect,* whilst membranous blastemata become pierced or loopholed tunics. This does not, however, prevent the blastema either from remaining amorphous or from undergoing fibrillation. Amongst the elementary granules, nuclei and cells, which occur in various number in solid blastema, it is more especially the two former, and *most* frequently the nuclei, that undergo further elaboration.

1. Even in recent fibrinous coagula, within the vascular system, rod-like *nucleus* formations are discoverable. They enter into the composition of many delicately fibred textures.

2. The nucleus is developed through the oblong form to the caudated nucleus, and from thence directly into nucleus fibre. Upon basement membranes, we often meet with serpentine, creeper-like nucleus-fibre stems. The caudated nuclei often constitute, when held together by an amorphous inter-cellular mass,—in rarer instances independently,—the fibrous element of not a few heterologous growths. More frequently, however, they enter singly into the composition of fibrous textures of other kinds.

3. Contiguous nuclei, in progress of fibrous development, conjoin and merge in the *varicose nucleus-fibre,* which by degrees acquires uniformity, and in rare instances forms the main component of fibrous new growths.

4. The nuclei form the basis of the *true elastic* splitting fibre.

Elementary granules forming in collateral array, become confluent, and establish, in various directions, more or less delicate, dark looking, longitudinal, or reticulated fibres, which resist the influence of acetic acid. They are most conspicuous upon basement membranes.

In solid blastemata the elaboration of cells into fibre occurs, for the most part, slowly and in the ordinary routine. The majority of the cells, however, remain undeveloped, and become reabsorbed. Still the development of primary cells into parent cells, however rare, does occasionally happen.

Fluid blastemata, in their development to textures, obey the laws of the cell theory (Schwann's). The perfect nucleated cell, however, originates in two different ways:

(*a.*) The union of several elementary granules gives rise to the nucleus, and around this to the cell, with the nucleus impinging upon the wall,—the ordinary mode. Or else—

(*b.*) The cell originates first,—its primitive limpid contents giving rise to an *endogenous nucleo-genesis*—for example, in the blood,—in exsudation—in colloid and medullary cancer.

Generally speaking, the nuclei equal in size those proper to physiological textures. Larger nuclei, however, and in particular oblong, free nuclei $\frac{1}{100}$th to $\frac{1}{50}$th of a millemeter in length, occur likewise,—in medullary cancer, for instance. Inclosed within cells, their further development, so far as we know, commences only after the conversion of the cell into fibre. They are round, oblong, lustrous, black-edged, or dull and granulated.

The cells present every variety of size, from that of the exsudation- and the pus-cell to that of the largest ganglion-cell, and upwards. They are in shape spherical, oval, lengthened by branch-like processes, rhomboidal, polyedrical.

They mostly contain one, often two, occasionally several (three, four, or five), nuclei.

The propagation of nuclei and cells occurs either immediately out of the fluid intercellular substance, as blastema, or within a parent-cell. Endogenous nuclei and cells [brood-nuclei and cells; filial cells; intra-utricular cell-formation] form within a primary cell, and distend it into a structureless vesicle, by the eventual bursting of which they become released.

In rarer instances, we meet with secondary cell-formation around a primary cell,—an *incasing of the primary* cell.

The primary cell is either permanent or adapted for ulterior development, namely—

1. The ordinary development of the cell into fibre. This is brought about by the spontaneous elongation of a cell to a wedge- or spindle-shaped, or a caudated cell; or by the fusion of several cells, arrayed in rows or columns, and engaged in the act of elongation to a varicose fibre, the protuberances of which are eventually reduced. Fibre produced in either way may, by splitting lengthwise, subsequently break up into fibrils. In form, the fibre corresponds with that of areolar tissue, or of organic muscle. The cell-nuclei immediately form into nucleus-fibre, into elastic fibre. In this wise do fluid blastemata, under the progressive consumption of the intercellular substance, give rise to fibrous new growths.

2. The above transformation differs from the working out of the primary cell into the *parent-cell,* and to the production of *pouch-like formations with endogenous nucleus and cell-development.*

(*a.*) The parent-cell is a *cyst-like dilatation of the primary cell,* and its contents furnish the blastema for the creation of filial cells, in either of the two modes before described. When the latter have greatly increased in number, the parent-cell frequently, but not invariably, bursts, and is destroyed. Not rarely, however, it becomes the groundwork for very remarkable textures. (See "Cyst.")

(*a.*) The *structureless parietes of the growing parent-cell acquire a fibrous texture,* and thus become fundamental to the type of the *alveolar* texture, and to cyst formation. (See "Cyst.")

(β.) The *parent-cell is singly*, or it may be in fusion with others, developed into a *gibbous, lobulated, hollow body, resembling a glandular acinus.*

The *filial-cells enter occasionally, even within the parent-cell, into a fibrous development.* Upon the dura mater, tumours are often met with seemingly of glandular texture. These consist of conglomerations of caudated cells, imbedded in a layer composed of the same elements. They are the product of a single parent-cell.

(*b.*) In fluid blastemata, utricular or pouch-like formations occur, similar to the tubular fibres mentioned under the head of solid blastemata, and they inclose nuclei and cells in various number. Their walls appear structureless; although, on a closer inspection, one or two nuclei,—occasionally several moveable nuclei,—may be detected upon them. They occur in colloid, in scirrhus, and in sarcoma, with a fluid inter-cellular substance. Their functional import is, in our opinion, identical with that of the parent-cell with its brood-elements. They present the greatest analogy with the capillary vessel and its contents, the more so that they probably originate through the fusion of nucleated or non-nucleated cells, arrayed in columnar juxtaposition. Their diameter ranges from the $\frac{1}{100}$th to the $\frac{1}{10}$th of a millemeter, and upwards.

We have now examined the essential elementary forms arising out of both solid and fluid blastemata. Their secondary arrangement into a texture offers equal diversity. Nuclei, cells, nay, elementary granules, display infinite variety in their arrangement, as do, in like manner, caudated nuclei and cells, and the different descriptions of fibres, in their course and in their coordination with other concurrent elements. These relations will have to be pointed out in the special analysis of new growths, to certain of which, peculiar arrangements naturally belong.

Other changes suffered by blastemata, either in their primitive state, or after having attained to different stages of development, are:

1. Resolution into a molecular point-mass. Blastema breaks up, in its primitive state, owing either to positive in-

trinsic relations, or to the absence of compulsory extrinsic conditions for its evolution. Or, again, it breaks up, after having already entered upon a course of development, owing to the cessation of the external conditions necessary to its maintenance and further elaboration.

In the state of disintegration, it may undergo complete or partial resorption, with or without entailing constitutional mischief. This process is often attended with cretaceous deposition, often with fatty conversion of the protein-substances.

2. The blastemata stop short at different stages, retrograde, and perish. This may happen at any epoch of their development, from the primitive state upwards. The causes may be either inherent in, or extrinsical to, the blastema. In some instances it is a natural death, certain elements, epidermis-cells, for instance, dying off, after having attained their highest development. This of course applies more especially to solid blastemata. In their primitive condition, they part with their water, condense, and shrink into horn-like masses, and frequently ossify. When more advanced in their development,—for example, to fibre—such elements waste, and become reduced to primitive amorphous blastema, which immediately shrivels, often disengaging calcareous salts, that is, ossifying. Within the cell there occurs incrustation, with amorphous granules (a kind of granule-cells), or in stratiform deposition. Here the blastema has become bereft of all faculty for further development.

3. *Conversion into fat*, occurs both in primitive blastema and in tissues, and it is frequently accompanied by the disengagement of salts of lime,—by cretefaction and ossification. The protein substances undergo a transformation into free fat in little molecules, and into cholesterine crystals. To this conversion both solid and liquid blastemata are liable. Where cells exist, it occurs in the shape of granule-cells.

Vogel describes it as a peculiar granule-cell development, established for the resorption of an inflammatory exsudation. "The exsudation," says he, "is converted into nucleated cells of $\frac{1}{300}$th to $\frac{1}{100}$th of a millemeter in diameter. These cells progressively enlarge, until they have attained the size of from $\frac{1}{80}$th to $\frac{1}{50}$th of a millemeter, and gradually fill, at first with a few, afterwards with very numerous little dark granules, until the cell, originally transparent and colourless, becomes thoroughly opaque, assuming the brownish or blackish coloration of its contents, and appearing as an aggregation of granules, which cover and conceal the cell-nucleus, and frequently even the cell's walls."

The concomitant chemical changes consist in the formation, or at least eduction, of a new (reckoning the cell's walls and the cell-nucleus,—of a third) substance within the granules, possessing the characters of fat, and occasionally of salts of lime. Vogel says, further on, "the matured granule-cells are not susceptible of ulterior organic development. After they have attained their full size, and filled with granule-cells, their further metamorphosis is a retrograde one. The cell-nuclei disappear, becoming, like the cell's walls, reabsorbed, whilst the granules, which alone remain, and are at first held together by a viscid medium, finally separate. After the complete breaking up of the granule-cells, the entire exsudate originally present is converted into a semi-fluid, pultaceous mass, which, with the aid of the microscope, is found to consist of, as yet, unchanged granules out of the broken granule-cells, natant in a fluid,—the original serum of the exsuded blood-plasma.

With reference to this process, which affects not alone inflammatory products, but every kind of blastema, we have additionally to state:

1. The process of granule-cell development consists not, we apprehend, in a development of fresh nucleated cells, and of granules within these. The granules become developed rather within the already existing cells, and also externally to them in the intercellular substance. There are seen distinct granules, which here and there collect in smaller or greater number, and occasionally assume an investment, not distinguishable from the bond-mass by which they are held together. Those developed within the cells accumulate and distend the cell's walls, until these give away and allow the granules to escape. This process may be directly witnessed, but it is further corroborated by the following circumstances:

(*a*.) Where the blastema is devoid of pre-existent cells, it does not contain any nucleated granule-cells either, but simply aggregates of granules; for example, in the fatty conversion of certain fibrinous coagula, of primitive muscle-fibrils, and of fibrous new-growths.

(*b.*) The exsudation-cell, the pus-cell, the cancer-cell, as the case may be, becomes the granule-cell, which retains the form of the pre-existent cell,—for example, the spherical, wedge-like, spindle-shaped, fibro-elongated granule-cell.

2. This process is, in point of fact, the fatty conversion of the contents of the cell. It is the counterpart of the fatty conversion of protein substances in every variety of blastema, and even in tissues generally. It gives rise to emulsive and saponaceous combinations, thus proving destructive to both blastemata and new-growths, which latter it would indeed render fitted for resorption, were this not often hindered by the simultaneous disengagement of phosphate of lime with cholesterine crystals.

These changes run parallel with chemical ones, consisting in the development of different kinds of gluten, in horny conversion, and the like.

3. Finally, blastemata (like physiological textures) become reabsorbed at various stages of development, having become adapted for the process by a previous disintegration or fatty conversion, although, in the case of fluid blastamata, without any intermediate change. Solid blastemata may become gradually dissolved and fitted for resorption by blood serum percolating the textures, for example, in solid, fibrinous, inflammatory products. Occasionally some of their nuclei are left behind, presenting the only visible residue of comparatively extensive blastema masses.

Our next enquiry concerns the conditions which favour the throwing out of pathological blastemata in particular localities. These may consist in an exsudatory process, not differing from that which presides over the normal act of nutrition, or else in processes which, though akin to physiological, are, in strict parlance, pathological. Such are hyperæmia, and inflammation in its numerous modifications. Again, blastemata become consolidated within the vascular system through the coagulation of fibrin, as metastases, or deposits.

HYPERÆMIA.

It is to be understood that we have to deal only with local hyperæmia—congestion, so called.

It consists in an excessive amount of blood in the capillaries of an organ; that is, in an injected condition of this latter, exceeding what experience has shown to be its average. This is not possible without dilatation of the vessels, nor can we admit the existence of congestion with coarctation of the vessels, and a *consequent* accelerated passage of the blood through them, which some have designated as active congestion.

A simple comprehensive view of the development of hyperæmia is not feasible, the conditions not being always the same. It will, perhaps, suffice to distinguish between *active*, *passive*, and *mechanical* hyperæmia. We shall here, however, not consider these individually—more especially, the two former—further than may be requisite for the establishment of a principle. Their closer investigation will be more appropriate under the head of inflammation.

1. *Active hyperæmia* is the result of external or internal stimuli acting immediately upon an organ, or reflected to it from other organs,—irritating the sensitive nerves, and thereby causing antagonistic palsy of the nervi vasorum, or, (according to another theory,) evoking an increased afflux of blood, a preternatural affinity of the parenchyma for the blood.

To this category belong, for the most part, those hyperæmiæ, dependent upon external or internal causes, which precede inflammations; most of those habitual, constitutional hyperæmiæ, rooted directly in the nervous system or in the blood; again, those hyperæmiæ occasioned by augmented activity, by over-wrought function, or concurrent with excited conditions of the organs.

2. *Passive Congestion* depends upon direct palsy of the nervi vasorum, wherewith is commonly associated a depressed energy in the remainder of the nervous system. The palsy may originate in the centres, or it may be peripheral. It is often determined by dyscrasis, and especially by the higher degrees of decomposition of the blood.

To this class must be referred those hyperæmiæ, introductory to so-called asthenic inflammations, in organs exhausted by excess of functional activity, enfeebled by hyperæmia and inflammation, or paralysed; as also in those hypostatic hyperæmiæ of the lungs, of the abdominal and pelvic organs, of the common integuments, which are developed under diminished impulse from the heart, in dependant parts of the body, during the progress of various adynamiæ and marasmi.

3. Mechanical hyperæmiæ, namely—

(*a.*) Hyperæmiæ arising from palpable mechanical impediments to the return of the blood through the veins, or to the ultimate disgorging of the venous trunks into the heart. The extension of the hyperæmia varies with the locality of the impediment. It affects single organs and sections of organs; for example, a portion of intestine strangulated, invaginated through tension and compression of its blood-vessels. Or it may have a more comprehensive range in impermeability of the liver, of the lungs, in stenosis (coarctation) of the heart's valves. The hyperæmiæ occasioned by spontaneous coagulation within the capillaries, by various elements obstructive of these vessels, as pus-corpuscle, cancer-cell, injected mercury globule, are commonly referred to the same head.

(*b.*) Hyperæmiæ ex vacuo, as they occur in atrophy of the brain within the unyielding skull, or in the gravid uterus, after rapid delivery, often to the extent of producing hemorrhage; hyperæmiæ due to the eccentrical rarifying atrophy of organs.

The hyperæmia is either of a more or less *transitory* or of an *abiding* nature, of which latter kind mechanical hyperæmiæ, from heart disease or from induration of the liver, present the most frequent and the most marked examples.

The sequelæ of hyperæmiæ are multiplex, varying with the duration, the repetition, the degree, of the congestion. Much likewise depends upon the character of the affected organ, the congestion being significant in proportion to the general importance of such organs, and to the vulnerability of its texture. Organs are prone to congestion proportionately to their vascularity and to the degree of their functional activity. Under particular circumstances of life, of occupation, of civilisation, certain organs, such as the brain and its membranes, and the lungs, are hardly ever entirely free from congestion. Hyperæmia affects morbid growths equally with normal formations.

Intense congestion suddenly developed in organs essential to life, (the brain or lungs,) may prove fatal directly, as so termed *vascular* apoplexy, or through the sudden effusion of blood serum into the textures—acute œdema.

High degrees of congestion occasion laceration of capillaries and parenchymatous hæmorrhage (apoplexy with bloody extravasation) in the brain, the lungs, and other organs.

The same causes lead, by an overloading of the blood-vessels, to absolute palsy of the blood-vessels, to stasis, inflammation, and gangrene.

Moderate but habitual or repeated congestion gradually engenders œdema and the dropsy of serous cavities—*genuine dropsy,* increased exsudation of blood plasma, preternatural nutrition of the textures—*hypertrophy, augmented secretion.*

In this relation, abiding mechanical hyperæmiæ, from heart disease, are worthy of especial notice, with their unfailing consequences, hypertrophy of the glandular abdominal viscera; preternatural secretion of the intestinal and bronchial mucous membrane; excessive, saturated secretion of bile.

Hyperæmiæ create and bequeath permanent dilatation and elongation with coil-like or serpentine deflection—properly termed varicosity—of the blood-vessels, as more particularly exemplified in the less resilient veins.

Hyperæmiæ frequently occasion and obviously accompany the development of various heterologous growths. Finally, in some organs, a proportion of blood pigment, effused with the plasma, constitutes the basis of rust-coloured, slate gray, bluish-black coloration, as in the lungs or on the intestinal mucous membrane.

Organs attacked by a high degree of hyperæmia present different shades of dark red, become swollen, loosened in texture, and consequently friable, lacerable. In organs of a porous, spongy texture, the swelling seems due to a bloated condition of the tissue itself.

HEMORRHAGE.

Hemorrhage consists in the extravasation of blood bodily, and in its entirety, from the blood-vessels, consequent upon a breach of their continuity. Herein it differs from red effusions resulting from the transudation through the parietes of vessels of blood serum, which, owing to various changes of admixture, has taken up blood pigment. Hemorrhage occurs either within textures, when it is, with reference to its attendant paralysis, somewhat inappropriately termed apoplexy; or else within natural or preternatural cavities and canals; for example, in serous sacs, in muco-membranous cavities or canals, pus-reservoirs, &c. The two kinds frequently coexist.

Heterologous growths are, in the same degree as normal formations, subject to hemorrhage, those at least which are highly vascular; as, for instance, adventitious membranes, carcinoma, (in a high degree,) the interior of cysts, &c.

Hemorrhage depends upon various causes, the most common cause being, as before stated, a breach of continuity in the blood-vessels.

Apart from hemorrhages produced by external injury inflicted upon blood-vessels, whether alone or in conjunction with other formations, those resulting from the following momenta, more especially capillary (parenchymatous) hemorrhages, possess a high degree of interest.

1. Hemorrhage the result of intense hyperæmia, of whatever kind. Thus, active hyperæmia has a marked tendency to create bronchial hemorrhage, passive hyperæmia uterine hemorrhages, hyperæmia from mechanical causes, in particular the bronchial and intestinal hemorrhages that result from heart disease, and the cerebral hemorrhages induced by a vacuum within the skull.

Finally, the excessive accumulation of blood determines rupture of the capillaries.

2. Another step conducts us to hemorrhage as occurring during the progress of inflammation, namely, in the stages of congestion and of stasis. In this combination we have hemorrhagic inflammation, and the in many respects remarkable exsudation designated *hemorrhagic*. Here, as in *simple* hyperæmia, the hemorrhage is unfailing, and considerable in proportion to the extent of the congestion and stasis, as also to the delicate, lax, and vulnerable nature of the implicated texture. Any one of these influences may predominate to a various extent. There are organs in which, owing to the nature of the texture, inflammation never takes place without

hemorrhage—for instance, the brain, the lungs, many heterologous formations, and especially exsudates undergoing a change of structure, and loose cancerous textures. The hemorrhage is capillary.

3. Hemorrhage from the laceration of vessels, produced in atrophied organs by the laxity and diminished resistance of surrounding textures—apoplexy of the decrepit uterus.

4. Hemorrhage from the spontaneous laceration of organs diseased in texture, pulpy and friable—laceration of the heart.

5. Hemorrhage from laceration of vessels consequent upon impaired texture of their coats, with or without dilatation of their calibre. It affects the smallest blood-vessels as readily as the main trunks, and more especially the arteries. Other coincident causes—hyperæmia, for example—greatly favour its occurrence.

6. Hemorrhage consequent upon the destruction of blood-vessels by ulceration, or by contact with a free acid, as in softening of the stomach.

It is questionable whether, and in what way, dyscrasial states can give rise to hemorrhage. A relaxation of the coats of blood-vessels sufficient to admit of the passage through them not only of plasma, but also of blood-corpuscles, is by no means proved, and its assumption, to explain the hæmorrhage occurring in scurvy or typhus, needless. The spontaneous hæmorrhage arising in the progress of such maladies, is the result either of local hyperæmia and inflammation, into which those general diseases have resolved themselves, or else of preternatural expansion (increased volume) of the blood itself, and of the consequent rupture of blood-vessels in the looser textures, such as the gums, the mucous membranes, and the lungs. Hence the occurrence of hæmorrhage in several organs simultaneously, and again the predominant invasion of a few particular organs. That hemorrhage, having once set in under such circumstances, is apt to become excessive, is due, without any doubt, to the slender coagulability of the dyscrasial blood.

Hæmorrhophilis—habitual hemorrhage—depends, so far as we at present know, upon a preternaturally delicate and vulnerable structure of the coats of the vessels, coupled with a thin, watery, condition of the blood.

Hemorrhage greatly varies in intensity. Its character is to be estimated not merely by the quantity of blood thrown out either externally or into internal cavities and canals, nor by the degree of anæmia that follows, but also, where the parenchyma of organs is its seat, by the anatomical condition in which we find the diseased parenchyma. In slight hemorrhage, the texture appears here and there dotted or streaked with extravasated blood,—*Capillary apoplexy*.—As the hemorrhage increases, these dots or streaks become more crowded, the parenchyma more turgid, until its interstices and cavities having at length become uniformly surcharged with blood, it appears throughout red. Or, where the blood has become coagulated, the rough texture seems as if converted into a blood placenta, whilst a diminished coherence of its molecules, and numberless lesions of continuity have rendered it friable, easily torn [mucous membrane, lung]. Such is the condition of the denser, more resisting textures; in those of a more lax nature, or where the hemorrhage has been sudden and violent, the texture is completely swamped and crushed into a red pulp of various shades, or else more or less laceration of texture has taken place, and the gap so occasioned become the recipient for the extravasated blood—*apoplectic foyer*.

The extravasated blood varies in deportment according as it is circumstanced subsequently to its extravasation. Certain influences cause it to undergo rapid and unusual changes; for example, in the stomach and intestines, black coloration and liquefaction of its fibrin, through the influence of gastric and enteric acid.

Blood poured out into cavities and canals, or into textures, is either fluid or in various phases of coagulation. Of these phases, coagulation with central or peripheral *encysting* separation of fibrine is the most important, owing to the increased impediment which it offers to the process of absorption.

The immediate effects of hemorrhage, besides the anæmia consequent upon great effusion, either out of the body, or into its cavities, are lesion of continuity in textures, in the shape of the swamping, crushing, or extensive laceration before referred to—impaired or destroyed function, paralysis of the organ affected—*cerebral, muscular hemorrhage*. A less immediate effect is the inflammation of surrounding textures, occasioned by actual injury, and by the irritation of the extravasated fluid, as a foreign body, with eventual organization of the products effused, callous condensation of the nether layer, and capsular isolation of the hemorrhagic clot. Inflammation, resulting in purulent and ichorus products, in parts broken up by hæmorrhage, is of rarer occurrence.

Hæmorrhage is both in itself, and in its results, of significance commensurate with the importance of the organ affected.

The cure of hemorrhage is a process simple or complex in a degree corresponding to the amount of blood effused, to its character as coagulable fluid, and to the extent of the injury suffered by the parenchyma.

Slight hæmorrhage is readily cured through resorption of the effused fluid, enabling the distended textures to recover their resiliency. The liberated red pigment, however, frequently resists absorption, even in slight hemorrhage, remaining strewn, for the most part in a state of minute molecular dispersion, over membranous formations, or between the elementary parts of a texture, as a brown or black pigment.

The remedial process is difficult and complicated proportionately to the amount of blood extravasated, to the resulting destruction of texture, and to the solidity of the coagulated fibrin as a central or a peripheral secretion from the fluid thrown out. It is a process of slow gradation, involving the changes produced not only in the effused blood with its red pigment and its fibrin, but also in the surrounding textures. These changes occur simultaneously, and we have frequent opportunities of observing them in areolar tissue, in muscle, and especially in the brain.

The crushed and disorganised texture within the walls of the foyer, together with the extravasate itself, undergoes liquefaction, at the same time that hyperæmia and stasis become developed in the adjacent texture-layer. The medium is probably almost exclusively supplied by the blood serum of the extravasated fluid which undergoes many changes in composition adapting it for the liquefaction of the different substances; namely, the solidified fibrin and the remaining elements proper to the effusion—blood-globules, nucleated and cell formations, *debris* of tissues. How important a part the liberation of adipose, and of saline substances out of their primitive combinations herein plays, is sufficiently attested by microscopic analysis. The blood-pigment incurs a special change. It is converted, partly within the blood corpuscles, partly extraneously to them, into a brown, rusty yellow, or into a blackish-brown, or a black pigment. This is shown as well in the form of spherical corpuscles, which resemble blood-globules and are frequently seen accumulated in compact congeries, as also in the shape of elementary granules (granulated pigment) either discrete or in circular groups. These are commonly free, but now and then inclosed within cells, or suspended from little prismatic crystals of ammonio-phosphate of magnesia. Together with them is found, at the part involved, fat in a free state, fat in the form of little black-edged, discrete or aggregate molecules, of limpid drops, of cholesterine crystals. Again, there are found elementary molecules down to the minutest pulverulent molecular mass, consisting of minutely subdivided, suspended fibrin, albumen, and fat, with calcareous salts. Finally, we have amorphous, membranaceous, stratiform coagula, nuclei, and blood discs (the as yet integral elements of the effusion), and amongst them all detritus of the involved texture.

In this manner the greater portion of the hemorrhagic effusion would have become fitted for resorption. The process is, however, impeded at this juncture, by the inflamed condition, and at a later period by the hardened character of surrounding textures.

Hence the ulterior metamorphosis of the effused mass, namely, its progressive thinning and clarifying into a mere pale buff, or, it may be, colourless liquid.

What, amongst other things, has become of the pigment? Partially it may have perished amid the unknown, final conversions of the fluid above characterised. To some extent, however, it is preliminarily taken up into a formation which,—derived from the coagulable contents of the effused fluid—invests the walls of the foyer.

This coloured, soft, jelly-like, loosely adherent lining, eventually becomes endowed with a minutely fibrillated structure, and even with blood-vessels, and is at length converted into a delicate, and, if the pigment be destroyed, into a colourless membrane, resembling a serous tunic.

Meanwhile, the inflammatory process has engendered in the walls of the foyer products which serve, in the shape of a nucleated blastema, (to be afterwards developed into fibrin texture of various kinds, and into areolar tissue,) to condense and harden the textures. Thus, the original hemorrhagic foyer is changed into a capsule or cyst, which, when it occurs in the cerebrum, is termed *apopletic cyst.*

This cyst is susceptible of diminution, and eventual closure, through resorption of its contents,—of closure to a cicatrix which often contains a certain kernel consisting of the aforesaid residuary pigment.

It must be confessed, however, that the complete closure of the cyst is a work of time and difficulty. This is intelligible from the slender absorbent faculty of the surrounding textures in their condensed and hardened condition. Occasionally, special obstacles stand in the way of this process of reduction; for example—

1. Great extent of the hemorrhagic foyer, and of the resulting cyst.

2. A vacuum, created either through original retraction, or through subsequent wasting of the texture involved, in muscles and especially in the cerebrum. In the brain, indeed, a later supplementary enlargement may take place in the apoplectic cyst, as an expletive of the vacuum created within the skull by consecutive atrophy of the cerebral organ; for the internal capsular membrane is, by reason of its vascularity, adapted alike for secretion and for absorption.

3. A very remarkable obstacle to the collapse and closure of the cyst consists in the secretion of fibrin in the shape either of central bulky, or else of peripheral isolating coagula, for the most part tinged by no inconsiderable proportion of embodied blood-corpuscles. These coagula, being originally very dense, and retaining their solidity even when converted into a fibrous texture, resist, when central, the liquefaction, when peripheral and encysting, the resorption of their contents.

Certain cases offer various exceptions to the processes hitherto described. Thus, some cysts having, in spite of the peripheral encysting coagula, parted with their blood-serum by early resorption, are found replete with a dark-coloured, inspissated, dry blood-plug; or else with fibrinous, stratiform, villous masses, developed out of the partially absorbed vehicle.

The frequency of hemorrhage varies greatly in the different textures of the organs. A scale of frequency is indeed but of very limited use, since hemorrhage is the result of various disturbances, and, in most instances, of the concurrence of several. Generally speaking, hemorrhages of the brain and of the bronchial mucous membrane are distinguished by their frequency; those of serous membranes are very rare, if we except the cerebral arachnoid sac.

It was signified at the outset, that the mere exsudation of coloured (red) serum, devoid of blood corpuscles, is perfectly distinct from hemorrhage. It is found as so called petechiæ (ecchymoses) in all textures, and in serous and mucous cavities as coloured effusion. It is due to decomposition of the blood.

ANÆMIA.

The chapter on hyperæmia naturally leads to a passing consideration of the opposite state, namely *anæmia.* Just as we have before treated only of *local* hyperæmia, we shall here, in like manner, limit ourselves to the subject of *local* anæmia. It comprehends both oligæmia, or an insufficient measure of blood in relation to what experience has shown to be its just standard, and true anæmia of an organ.

It is present under various conditions:

1. As the partial manifestation of general anæmia.
2. As the consequence of hyperæmia of one or more other organs.
3. As the result of coarctation and closure, or orificial obstruction of the vascular trunk supplying the diseased organ or part, pending the establishment of a compensating collateral circulation.
4. As the effect of external or internal pressure upon an organ, and its consequent inadequate injection; anæmia of the lungs from pleuritic effusion; anæmia of textures the interstices of which are filled up with morbid products, as in hepatisation of the lungs, in fatty infiltration of the liver.
5. As a consequence of decay of the vascular apparatus of an organ affected with atrophy, whether primary or secondary, more especially atrophy with condensation (concentrical wasting).

The effects of anæmia are pallor, collapse, and shrivelling of the textures, weakening and eventual extinction of their function.

Anæmia is momentous proportionately to the vital importance of the diseased organ, and to its exigences with respect to the supply of blood. Thus, anæmia of the brain, of the lungs, of muscle, is of the highest import.

INFLAMMATION, PHLOGOSIS.

This pathological process is of paramount interest, not only on account of its great frequency, and of the great variety of external causes by which it is called forth, but also as being that in which most, and in a certain sense all, general diseases become localized. It is a process which leads incontinently to the most various and most extensive new-growths, and associates itself, equally often, to other anomalous formative efforts. In fine, it is a process which, on the one hand productive, on the other hand frequently proves destructive of both normal and anomalous formations.

The inflammatory process is capable of being experimentally called forth and observed, in all its phases, in transparent textures. Thus studied, it has furnished the groundwork for the most varied interpretations, but, at the same time, for researches respecting other exsudatory processes. Nor have these experiments failed in a certain measure to elucidate the connexion that prevails between blastema and the endogenous formative processes carried on in the blood itself.

We seek not to deprive this process of its time honoured name, *inflammation*, because it has become naturalized in science beyond all others. It is applicable enough, if in using it we simply dismiss the theories which first led to its adoption. It comprehends the entire process, and the efforts made to designate the latter differently have utterly failed. Andral's hyperæmia, and Eisenmann's stasis have not advanced the subject by a single step.

It is impossible to define inflammation suitably, owing, on the one side, to our imperfect knowledge of its proximate causes, and, on the other, to the complex nature of its consecutive phenomena. These latter, variously modified in type, point to processes equally varied, whilst they at the same time furnish the most striking analogies with other processes which issue in exsudation (production of blastema).

Let us now proceed to a descriptive examination of the phenomena which constitute so many stages of the process, a due regard being had to the results of experiment, that is, to the observation of the inflammatory process as artificially called forth in animals. And in this description, together with the analytical remarks annexed to it, we will take for our basis the so called pure, legitimate, inflammation, which yields essentially a coagulable, fibrinous, plastic product, as developed in sound organisms without the cooperation of a pre-existent dyscrasis, and simply as a consequence of moderate local stimulation; such being the most marked of any in its manifestations and stages.

The phenomena of the inflammatory process present the following sequence.

1. The moderate influence of mechanical or chemical stimuli is followed by contraction of *the capillaries, and simultaneous quickening of the blood-stream through them.* This phenomenon may be wanting as an effect of most causes of inflammation in the human species; and even in experiments upon animals it is either transitory or entirely absent if the stimuli applied be potent.

Contraction of the vessels is succeeded sooner or later by—

2. *Dilatation of the capillaries,* if this be not, indeed, the very first cognizable phenomenon. Unlike contraction, it is invariably present, readily seen both in the living animal and in the dead subject, as is, in like manner, the simultaneous loading of the vessels with an increase of blood. It determines capillary injection, and therefore the redness of injection proper to inflamed textures.

This dilatation of the vessels is attended by a *retarding of the blood-stream,* which sooner or later, although not always visibly, merges in an oscillating movement of the blood in the capillaries. The contained blood-columns move forward and backward by turns, the onward movement, however, predominating. The blood-corpuscles begin to adhere to one another, like rolls of coins, the outer linear layer of plasma (the lymph space) within the vessel still remaining unchanged.

This twofold proceeding establishes the stage of congestion.

3. Hereupon ensues, sometimes so rapidly as to prevent the retardment of the blood-stream and its oscillation from being noticed, *stagnation of the blood-stream,—stasis.* The blood-vessels are completely filled up with blood-corpuscles, so that the transparent, so termed, lymph space, near the circumference of the vessel and before occupied by plasma, has vanished. Meanwhile the blood-corpuscles have assumed greater intensity of colour, have become flattened, contracted, and firmly glued to each other, and to the vessel's walls, so as to form a homogeneous red mass, with irregular translucent intervals.

The nuclei and nucleated cells (so-called lymph globules and colourless blood-corpuscles) have increased in number to an extraordinary extent, often adhering together in groups connected by delicate transparent coagula, and forming either thus, or singly, the aforesaid translucent intervals. The blood has assumed a dark tile-coloured aspect, verging upon cherry-red.

In the preceding stage (see hyperæmia), as well as in this, two notable phenomena are witnessed, namely:

(*a.*) Laceration of blood-vessels, and extravasation into the textures, or into the free spaces,—lung-cells; muco-membranous cavities and canals; serous sacs. The hemorrhage is frequent and considerable proportionately to the degree of congestion and stasis, and also to the delicacy and the textural looseness of the diseased part. For the most part it takes the form of capillary apoplexy, and only in very delicate normal and anomalous textures, as, for example, the brain or encephaloid cancer, the form of the isolated clot.

(*b.*) Transudation of blood-serum through the thinned blood-vessel walls into the parenchyma, and from membranous expansions into the cavities and canals of which they form the lining. This implies, in the parenchymata, pervading moisture,—in expansive structures, diffuse exsudation or circumscribed accumulations beneath the epidermis,—for example, in burns, in vesication artificially called forth, in erysipelas, and the like. The exsuded serum resembles essentially blood-serum, only that it is, for the most part, less rich in albumen. This phenomenon often merges at once in the exsudatory process next succeeding, the two acts being simultaneous.

4. The step to which stasis ultimately leads is *genuine effusion*, that is, the exsudation of blood-plasma, a fluid holding in solution fibrin, albumen, and salts. It is thrown out into parenchymata, filling up their interstices to a various extent, either as a fluid, or as a more or less solidified product. Or, again, it is partially, or, it may be, wholly expended upon the free surfaces of natural cavities and canals, or of such as have arisen out of the previous transudation of serum, for example, in vesication of the epidermis, or in antecedent suppuration of textures,—in abscess cavities.

With exsudation, the inflammatory process is to be looked upon as closed. It is immediately followed by an endosmotic current of the serous portion of the effusion, causing the blood-corpuscles to float in a thinner medium, to exchange their now flattened for a more spherical shape, to become separated, to part with a portion of their pigment, and finally, by dint of a returning resilience in the blood-vessels, to move, conjointly with the aforesaid form-elements, onward again in the circulating stream.

We have now furnished the reader with a substantial description of the several acts which make up the process of inflammation. We shall further endeavour to show how these may be reconciled with the present standard of pathological science.

1. The first experimental phenomenon adverted to, namely, contraction of the capillaries, with acceleration of the blood stream, has been stated to be inconstant, and indeed absent altogether, when potent stimuli have been used in the first instance. Where it does occur, it is to be regarded as a vital phenomenon. The contraction of the blood-vessels is moreover independent of any simultaneous collapse of the parenchyma, like that produced, for example, by the action of cold.

2. The dilatation of blood-vessels and the retardment of the blood-stream are, on the other hand, constant and essential. The very fact of their being so commonly the primitive phenomena, or at any rate of their succeeding very rapidly to a previous contraction, refutes the notion of their consisting in a secondary relaxation, resulting from exhaustion. With these, and with the subsequent stasis, the theories of inflammation hitherto advanced are mainly concerned.

Henle reduces these to an attraction-theory, and to a neuropathological theory.

(*a.*) The attraction-theory refers the essential phenomena of inflammation to an augmented affinity between the parenchyma and the blood, and especially to an anormal attraction of the blood, and of the blood-corpuscles in particular, by the affected parenchyma. It assumes the retardation of the blood-stream, and the crowding together of the blood-discs to be the primary—the dilatation of the capillaries a secondary phenomenon. It explains even the stasis as a continued increment of that attraction.

The augmented attraction is effected through the intervention of the nerves, and either by direct influence upon the peripheral nerves, or else by reflection from the centres of the nervous system.

The attraction-theory carries the problem of congestion and stasis a step farther, without solving it. Together with its common attribute, namely, an increased afflux of blood to the diseased part, as due to a dilatation, and to a more frequent contraction of the afferent artery, it has been met by many valid objections. It is only the stasis and inflammation engendered by a pre-existent dyscrasis that can, provided the attraction be not limited to the blood-corpuscles, but embrace the diseased plasma, warrant the conclusion of a preternaturally strong affinity between blood and parenchyma. There would be some analogy between such a localization of general disease, and the indwelling relation of secreting parenchymata to certain normal or anomalous ingredients of the blood. But these inflammations, like the rest, are aptly expounded in the neuropathological theory.

The neuropathological theory, on the contrary, assigns to the nerves an important part, and ascribes the accumulation of the blood to the dilatation of the blood-vessels, this being set down as the primary—that as the consecutive phenomenon.

The dilatation of the vessels is the consequence of paralysis of the nerves. Respecting the cause and the conditions of this paralysis, there exist two different opinions.

According to Henle, an antagonistic relation prevails between the states of irritation of the sensitive nerves, and of the nerves of the blood-vessels: a high degree of inflammatory irritation in a sensitive nerve producing a state of depression—in a word, paralysis of the implicated blood-vessel nerves.

According to Stilling, the sensitive and the blood-vessel nerves bear, on the contrary, a direct sympathetic relation to each other. He assumes a continual reflex action to be kept up by the sensitive, upon the blood-vessel nerves, whereby the tone of the latter is sustained. With paralysis of the former, the tone of the latter is destroyed; whereas by excitation of the former their reflective power is augmented, and the tone of the latter thereby raised. In accordance with this reciprocity, two different kinds of inflammation are made out.

Griesinger was led to the adoption of a similar view, imagining pain to result from a qualitative disturbance of the texture of the nerve.

Against this hypothesis it is to be objected that a continued reflex action of the nerves of sensation upon those of the blood-vessels as a necessary condition for the undisturbed function of those vessels is not proved; and that the assumption of two different characters of inflammation, involves both a contraditio in adjecto, and a disregard of the results of observation. An inflammation with augmented tone of the blood-vessels cannot exist, and this admitted, inflammation with and through paralysis (diminished tone), must invariably ensue from influences paralysing to the sensitive nerves. This is, however, opposed to daily experience.

Our own opinions accord with those of Henle, whose theory we shall therefore adopt as the ground work of any future remarks on this subject.

Even here the causal momentum influences the peripheral nerves, producing either at the spot itself, or through the intervention of the nervous centres, in other sympathetically allied structures, excitation and depression. Or again, the influence affects the nerves within the centres, the impression being conveyed from *thence* to the corresponding peripheral organs.

The stasis is not accounted for in the neuropathological theory. It is, indeed,—

3. Not intelligible upon the ground of paralysis and dilatation of the blood-vessels, even though [as we must admit a certain off-flowing to take place to the veins] we may not regard

the stasis as an absolute one. For our own part, we hold stasis to be dependent upon the following momenta:

(*a*.) The cohering, crowding, and impaction of the blood-discs within the capillaries, the blood-plasma being partly withdrawn into the veins.

(*b*.) The thickening of the plasma, and its saturation with fibrin and albumen, owing to the transuding of blood-serum through the distended and thinned blood-vessel walls.

(*c*.) The accumulation of the colourless globules—that is, nucleus and cell-formations—along with the blood-corpuscles, their conglutination, and the delicate transparent fibrinous coagula collaterally developed. This is, perhaps, the most important stage in the inflammatory process, as at once illustrating the stasis itself, and embracing the plastic processes in which the blood engages when arrived at this point. A line of distinction is thus drawn between the inflammatory process, and a simple process of exsudation. The form-elements adverted to are not merely washed together within the range of the stasis; they are new creations out of the blood so arrested, which at the same time undergoes other remarkable changes. Thus it is of a dark red, with a tile-coloured shade, contains red flocculent particles of cruor, visible to the naked eye, teems with the aforesaid elementary bodies, and with coagula, most of which latter have incorporated a number of the former, as well as of dark coloured flattened blood-corpuscles. The accumulation as well as the general importance of those (new) elements, for the inflammatory process, and especially the stasis, have been recognised by Addison and others.

The momentous question, as to the cause of the said formative process of blood in the condition of stasis, will be answered, so far as it is possible, in the sequel.

4. *Exsudation.*—The thinning and permeability of the walls of blood-vessels, produced by their distension, must be regarded as the basis of this phenomenon, even in the instance of a condensed plasma; perhaps, additionally, the effort at equalization betwixt the latter and any thinner blood-serum before exsuded.

A question of peculiar interest here suggests itself, namely: wherefore in the inflammation of membranous expansions does exsudation always take place upon their surface, and into the cavities which they invest, whilst the effusion beneath is limited to the infiltration of the implicated parenchyma, or of the subjacent areolar tissue—with, for the most part, an inconsiderable amount of plastic serum? This applies not only to mucous and serous membranes, but even to other more delicate hollow bodies—for example, follicles. The problem, like that of Johannes Müller, as to natural secretions affecting the free surfaces, is only to be solved upon the ground of less resistance being offered in this direction. The said infiltration of parenchymata and of contiguous textures, is co-significant with the œdema that surrounds patches of inflammation.

To an indefinite distance beyond the range of true inflammation, and lessening in intensity as the distance increases, congestion takes place, and with it the effusion of serum. And this serum becomes, in like manner, poorer in plastic substances towards its periphery. Such is *inflammatory œdema; œdema encompassing the range of inflammation.*

The accompaniments of *pain, redness, heat, swelling,* are explicable as follows:

Pain is determined—

1. In external injuries, as wounds, burns, cauterisation, either by the immediate action of the cause of the inflammation upon the peripheral nerves, or else by reflection from the central organs. In no instance is it determined by the inflammation *itself.*

2. By the pressure and tension which the dilated and overladen vessels, and the effused fluid exercise upon the nerves, *true inflammatory pain.*

3. Finally, pain of a certain degree, or rather of particular kinds, is to be referred to augmented temperature in an inflamed part. In the absence of increased warmth, which characterises certain inflammations, pain is generally absent, also.

The redness is a consequence of the overloading of the dilated capillaries with blood-corpuscles; it is therefore to be designated as the *redness of injection.* A new creation of blood-vessels does not, as was once supposed, ever accompany the inflammatory process itself, and cannot, therefore, be taken into account here.

The *redness* is also, in some measure, due to the blood thrown out during the stage of congestion and stasis.

In some inflammations, one great source of the redness is the drenching of the tissues with dissolved blood pigment,—*redness of imbibition.*

In form, the redness of injection varies in different textures, according to the order in which their capillaries are disposed. Take for example the linear redness of injection in inflamed fibrous structures. In the most vascular structures, however, the naked eye is no longer cognizant of aught but a uniform red tint.

Lastly, the redness is subject to many gradations of colour, being deeper in proportion as the organ is vascular, and the congestion intense. Much depends, moreover, upon the constitution of the blood, and more especially of its red pigment; take, for instances, the copper-redness of the syphilitic, the violet hue of the typhous stasis. The *elevated temperature* has its source partly in the formative processes, in which blood in the condition of stasis becomes engaged, but for the most part in the excitation produced upon the sensitive nerves.

The swelling is dependent—

(*a*.) Upon dilatation and repletion of the capillaries;

(*b*.) More especially upon exsudation of blood-serum and plasma;

(*c*.) Upon concurrent hemorrhagic effusion (extravasation).

The two latter conditions give rise, in like manner, to the loose, lacerable condition of inflamed textures.

After this description of the inflammatory process, and this interpretation of its phenomena, we should proceed to investigate the so-called issues of inflammation. These, however, resolution excepted, cannot be satisfactorily considered without an insight into the various products of inflammation, nor these products themselves be rendered intelligible but by a knowledge of the manifold varieties and anomalies of the process, and of its relation to the blood-crasis.

VARIETIES OF INFLAMMATION.

Inflammation is characterised by much variety and anomaly. On the one side, it recedes so far from the foregoing description of the process, that it has been attempted to distinguish certain forms as *spurious inflammation*. On the other side, its gradations into mere hyperæmia and a preternatural amount of plasma-exsudation (nutritive irritation) are so imperceptible, that discrimination becomes a matter of difficulty, the greater, perhaps, that such processes frequently do become exalted into, and do alternate with, inflammation.

Every hyperæmia may attain the point of inflammatory stasis.

(*a.*) *Active hyperæmia,* the same in origin with inflammatory congestion, is developed into *sthenic,* or active, *inflammation.*

(*b.*) *Passive hyperæmia,*—the result of direct, centrical or peripheral palsy of the entire nerve apparatus of an organ,—becomes *passive, asthenic inflammation.* To it belong, amongst others, all hypostatic inflammations occurring in dependent parts during the progress of adynamiæ and marasmi, and the asthenic inflammations in organs paralysed by concussion, complex injuries, or by direct central influence,—for example, of the bladder, in paraphlegia. Many of them have a humoral origin, a dyscrasial disturbance giving rise to paralysis either central or directly peripheral. The hyperæmia and stasis are characterised, in the absence of pain and increased temperature, by very dark livid redness, partly of injection, but for the most part of imbibition. Their products, conformably with the humoral elements, are poor in coagulable materials, discoloured by adherent blood-pigment, spuriously reddened, sero-albuminous, sero-purulent. Frequently the stasis becomes absolute, degenerating into necrosis of the blood and of the diseased texture; in one word, into gangrene.

(*c.*) *Mechanical hyperæmia,* as we have seen, commonly determines exsudation of serum [œdema], and this not alone from the true capillaries, but also from the larger veins. By intense mechanical obstruction, the hyperæmia is raised to a stasis marked by very deep redness, great tumefaction, numerous lacerations of vessels, and hemorrhage. It deposits the usual coagulable products, but often degenerates, with complete paralysis of the organ affected, into absolute stasis and gangrene.

The course of inflammation is *acute* or *chronic;* all else being equal, sthenic, and traumatic inflammations, and (amongst those due to internal causes) such as result from a fibrino-croupous crasis are marked by their acute character. Asthenic inflammations, on the other side, and of these, more especially the ordinary hypostatic, incline to a chronic course. *Chronic inflammation* is variously modified.

(*a.*) Inflammation may tarry unwontedly long at any one of its stages. It is in unison with the causal conditions upon which hypostatic inflammations depend, that their stasis should be abiding. The congestion is, moreover, often only very gradually brought about.

(*b.*) There are inflammations in which a decided stasis is most probably never arrived at; the process consisting in prolonged congestion, with a slackening of the circulation, bordering upon stasis. Arrested processes of this kind give rise to exsudations, poor in coagulable materials, serous in kind, and prone to accumulate either by slow degrees or by sudden impulses.

(*c.*) Chronic inflammation consists in a linked succession, stage for stage, of inflammations more or less acute. Amongst the most marked are those occurring in the vascularized products of an antecedent inflammatory process, more particularly in the pseudo-membranous products of inflammation upon serous membranes.

(*d.*) Inflammations productive of pus and ichor, whether of external or internal origin, are, for the most part, essentially chronic processes imitative of a secretive function.

In point of degree and extension, inflammations offer the greatest possible variety. For the degree of intensity of the inflammation, the intensity of the external appearances, especially redness and tumefaction, together with the quantity and quality of the products, affords a criterion. This, however, applies exclusively to inflammations due to local, external causes. In inflammations kindled out of an internal humoral element, the mildness of the stasis and of its symptoms is often strikingly disproportionate to the quantity and character of its products. Even the former kind is subject to frequent exceptions: thus, purulent exsudation, which is commonly regarded as the product of a very intense stasis, not unfrequently occurs in great abundance under slight symptoms.

The extent of an inflammation, that is, its diffusion over one or more organs, depends both upon its occasional cause and upon accessory circumstances. Inflammations evoked by dyscrasial agency, very commonly assault large organs, or at least considerable sections thereof, very frequently several like-named or kindred organs, at once, or in rapid succession, —for example, serous, mucous, follicular formations.

Inflammation is not essentially modified by anatomical differences of organs and tissues. We may here, however, remark that—

(*a.*) Organs differ greatly in their proneness to inflammation from an internal humoral cause, there being a certain relation of organs to special crases. Frequently, however, peculiar extraneous impulses concur to localize a crasis upon a particular formation; take, for instance, the localization of the puerperal crasis upon the peritonæum, a part enlisted both in the act of parturition and in the puerperal uterine process.

(*b.*) Vascularized new-growths, like normal textures, may become the seat of inflammation, luxuriate in growth, and increase in substance, or else decline and perish. Sloughing and luxuriance of growth are here occasionally concurrent processes. Tender, budding formations are especially apt to become destroyed by inflammation, at the same time that a more vigorous germination is becoming established in their vicinity.

The distinctive characters of regeneration and of suppuration, destructive inflammation, will be considered under the head of terminations or issues.

Both the causes and the products of inflammation constitute very important grounds of distinction, and a rigid discrimination is necessary between inflammations from external, and inflammations from internal causes. To the former class belong not alone the inflammations locally engendered by direct external influences, but also those awakened in remote but sympathetically allied formations, through the mediation of the nerves. To the latter class belong those hyperæmiæ and stases based upon a greatly overwrought condition of the nervous centres, be it irritation or paralysis, and more particularly inflammations arising out of a pre-existent dyscrasial or humoral element.

RELATION OF THE INFLAMMATORY PROCESS TO CRASIS.

A twofold relation exists between inflammation and an anomalous crasis, the latter being either a mere result of the inflammatory process and secondary, or else pre-existent and primary, and the inflammation a consecutive, symptomatic phenomenon,—the localization of the crasis.

1. The undeniable development of a dyscrasial condition of the blood, as the sequel to mere local inflammation produced by external and, it may be, traumatic influences, justifies the inference that inflammation is in itself a dyscrasial process. All doubt as to this point is, however, overcome if we consider—

(*a.*) The qualitative variations in the products of a local inflammation; and still more—

(*b.*) The fact that, where the blood is in the condition of stasis, the elements of the exsudation are found preformed within the vessels. The character of the products is thus shown to be mainly dependent upon inbred transformations of the stagnant blood, and more particularly of its plasma.

Inflammatory stasis is thus shown to be no mere passive congestion; effusion to be no mere percolation of an altered plasma simply divorced from the blood-globules. On the contrary, both are shown to imply every variety of conversion, from simple exaltation of the formative process carried on in normal plasma, to the generation of totally heterogeneous combinations and of corresponding form-elements. For the same reason the plasma effused, and the fibrinous matter thrown out as the result of inflammatory stasis, differ from mere extravasated plasma, or extravasated fibrin. The same arguments, in fine, serve for the solution of the question: in what wise does an anomalous constitution of the entire blood-mass result from local inflammation?

Consecutive dyscrasy, as infection of the blood, is brought about—

(*a.*) Through direct readmission of effused matter into the circulation by endosmosis, or through the indirect way of resorption through lymphatics;

(*b.*) Through a more copious reception of the products of inflammation into blood-vessels laid open by external injury, or by ulceration and necrosis;

(*c.*) And (besides these commonly-taught channels) through the ebbing back to the roots of the veins of the altered plasma, issuing from blood-vessels involved in the process of inflammation.

This last mode of infection is decidedly the most common. It is, indeed, the only one possible where endosmosis and resorption of the substances effused are, owing to the density of the latter surpassing that of the lymph or of the blood, excluded. It resembles, on a small scale, that infection of the blood due to inflammatory products directly thrown out into the cavities and canals of the circulation from their internal membranes.

The interest and the practical importance of the subject warrant us, whilst referring to the chapter on blood diseases and on pyæmia, in remarking here that,—if we except cases engendered by very heterógeneous substances, the result of decomposition within or without the vessels,—infection stands in direct relation to the magnitude and number of the inflamed parts, and, as a consequence, to the amount of inflammatory products taken up into the blood. In opposition to a fermentation theory, we may affirm that a minimum of inflammatory product does *not* suffice to determine a perceptible alteration of its admixture, very small quantities merging in the normal processes of the circulating mass. The most pregnant source of infection is the inflammation of the internal membrane of blood-vessels.

The *character* of the infection and of the consequent crasis corresponds with that of the products of the inflammatory stasis.

Consummated infection of the blood-mass bears towards every inflammation which it may subsequently call forth, the relation of a primitive crasis localized in congenial inflammation.

2. In every crasis, hyperæmia and stasis may occur accidentally. Here, however, we are more particularly concerned with inflammations resulting from a special crasis as their internal and sole cause. To this class belong a multiplicity of anomalous crases, more especially, however, the fibrin-crases and pyæmia, the former being usually comprised under the so-called phlogistic crasis. Inflammations dependent upon dyscrasial impulses are marked by their preference for particular organs, and, for the most part, by the acuteness of their career, as well as by the rapid formation of their products.

This last circumstance is, to a certain extent, practically demonstrable with respect to the fibrin-crases and pyæmia. In these, the examination of the blood shows that, the same changes occur in its totality, as in any portion of it in the condition of stasis; that both the plasma and the fibrin are constituted identically with the products of the stasis. Herein a source is supplied for the rapid development of the stasis, and, consequently, of the effusion. In the stasis, a simple increase of the processes carried on in the totality of the blood suffices to furnish forth, in exquisite form, a characteristic exsudation.

Inflammation of humoral origin is determined, according to the neuro-pathological theory, by antagonistic palsy of the nerves of the blood-vessels, brought about, at the periphery or at the centres, by the irritating effect of the dyscrasial blood upon the sensitive nerves; or, if passive, by direct paralysis. To such inflammations unquestionably belong many commonly ascribed to an external cause; for example, pneumoniæ. These we believe to be, for the most part, the localization of a crasis modified by atmospherical influences.

The relation of the crasis to particular domains of the nervous system, determines the localization of certain crases in particular organs, almost as a definite rule. Still, the modifying power of concurrent external influences is not to be lost sight of.

Inflammation arising out of humoral elements manifests itself not unfrequently as a metastasis, exhausting the crasis and resolving itself into a local evil.

The above becomes invested with greater significance when applied to the exsudatory processes upon which the production of various heterologous formations rests.

EXSUDATION.

The greatest and most marked differences of inflammation are manifested in its products. In inflammations due to a preexistent crasis, such difference is intelligible enough, the product of the stasis having its germs, in part at least, preformed in the general circulation. It is less clear with respect to inflammations devoid of a pre-existent crasis. Mere local inflammation, however, independent of any dyscrasial materials, will yield products presenting a repetition of many, if not of all, the characters which mark the products of dyscrasial inflammations, such as simple (plastic) fibrinous exsudation, fibrino-croupous (pyin-holding) exsudation, pus, ichor. Here the question is: wherein are these different characters founded? how elicited by stasis out of normal matter? Their ultimate source can be no other than the stasis itself, of which it might be predicated—

(*a.*) As stasis it promotes, and, within its range, condenses upon inconsiderable elementary materials, the changes and formative processes which take place in the general circulation.

(*b.*) When feeble in intensity it determines mechanically the display of thin, serous, sero-fibrinous, sero-albuminous substances, poor in plastic materials.

(*c.*) Greater intensity and duration occasion relatively, through the continued afflux of oxygen to a given quantity of plasma engaged in stasis, excessive fibrin formation, and ulterior oxydation of the fibrin. Hence result fibrinous exsudation, and, as a higher degree of oxydation of the protein (Mulder), croupous constitution of the fibrin, croupous exsudates, pus- and ichor-exsudates.

(*d.*) *Absolute stasis*, deficiency of oxygen, leads to necrosis of the blood and of the implicated textures,—*gangrene.*

The exsudate is, accordingly, now of general, now of local import. Exsudates, when formed, suffer various changes through external influences, and take various impressions not inherently theirs; thus not unfrequently entering upon anomalous modes of development. Like blastemata, they do not exsude unalloyed; croupous fibrin and pus, for example, being always mingled with a certain amount of so called plastic fibrin and normal plasma. Nevertheless, the study of exsudates in their utmost simplicity of forms is indispensable.

Exsudates, indeed, both in their primitive condition, and in the changes which they undergo, irrespectively of impressions from without, afford an insight into the local processes occurring in blood involved in stasis, as also into the processes carried on in the general circulation, that is to say, into the crasis itself.

The most instructive of all exsudates are those occurring upon serous and mucous membranes, upon which many, if not most, crases are wont to localize.

The doctrine of inflammatory exsudation is, in many points, applicable to the products of other processes of exsudation, nor shall we, in the sequel, neglect the opportunity of generalizing in this sense.

Let us pass, in the meantime, to the enumeration of exsudates as established according to anatomical research, taking at the same time, a general survey of the various changes which they undergo.

1. *Fibrinous exsudation* comprises several varieties, each corresponding to a particular constitution of the fibrin of blood involved in stasis, or of the general circulation. They are, for the most part, not pure, one kind being always alloyed with a proportion, however minute, of another kind, and a certain amount of normally constituted fibrin attaching to all.

(*a.*) *Simple or plastic fibrinous exsudation.*—A grayish, yellowish gray, or if containing blood-corpuscles, even red, reddish gray exsudate which, in great part, speedily solidifies, into bulky, membranaceous, plug-like, or frame-like coagula, whilst the remainder, where the proportion of blood-serum is considerable, coagulates in the latter into flocculi, thus parting into a solid and a fluid constituent. It presents itself, on a closer inspection, as a clodded, fibrous, diaphonous blastema of very tenacious properties, the broken surface of which is felt-like and studded with nuclei and nucleated cells. It answers to fibrin 2. It is observed in its purest form in wounds, when these agglutinate and heal by the first intention. Besides this,* it occurs upon the pleura, in areolar tissue, in muscles, in bones, upon the endocardium and the internal blood-vessel membrane, in the brain, occasionally, as pneumonia, in the parenchyma of the lung, which either slowly recovers its natural state through absorption, or ends in induration with extinction of the pulmonary texture. Upon serous membranes it constitutes the peripherous coagula lining the inner surface of serous sacs. It frequently enters, in common with a certain proportion of normally constituted fibrin, into the composition of other exsudates, as that portion of them upon which a change of texture depends, for example, into the croupous, the purulent, &c.

The following are its metamorphoses, the most remarkable amongst which is its textural conversion.

(*a.*) It becomes partially or wholly reabsorbed; this occurs slowly, through the instrumentality partly of the serous portion of the exsudation itself, partly of a supplementary effusion of serum, succeeding to the resolution of the stasis. These humours furnish the solvent—the corroding media, so to speak, for the solidified fibrin effused, having incorporated which, layer by layer, either in solution, or in a state of minute subdivision, they forthwith become reabsorbed. In like manner, the fibrinous coagula within the vascular system; for example, vegetations, thrombi, &c., are progressively taken up again into the blood.

(β.) It wastes and hardens, with the loss of its fluid part and with lessening of its volume, to a horny, and eventually, perhaps, to an ossified mass.

(γ.) It undergoes a change of texture, commonly, and for the greater part, consisting in an ulterior development of that fibrillation of the blastema which ensues upon the solidifying of the exsudate. To avoid repetition, we refer for the details of this process to the heads—new growth of areolar tissue, and fibroid formations.

In rare instances, the new formation of blood-vessels is predominant therein, more especially in exsudates upon the arachnoid membrane. In the vicinity of serous membranes, there result serous layers, a new serous sac; in contiguity with bone, bony texture.

The time requisite for these transformations is scarcely defineable in a general way. Under favorable circumstances, they are wont, even in voluminous exsudates, to attain an advanced or nearly completed state, within the space of six weeks.

The constitution of the exsudate corresponds, as before stated, with that of fibrin 2, having either been acquired in the stasis or pre-existed as a blood crase. It is a quantitative anomaly consisting in an exaggeration of the formative processes which occur in normal plasma,—and especially in the locally and generally increased generation of a fibrin (hyperinosis) marked by its coagulable and plastic properties.

(*b.*) *Croupous exsudation* has several varieties, dependent upon a qualitative impairment of the fibrin. It is, with the utmost impropriety, confounded with the former and its kindred crases. It is marked by a high degree of coagulability; by a yellow, or greenish yellow coloration; by its opacity; by its inorganisable nature; by its early tendency to break down, to liquefy; frequently by a corrosive, texture-softening power. The quantity of serum simultaneously thrown out is relatively insignificant.

The croupous process of exsudation and its product are further distinguished.

1. By the commonly excessive, exhausting, quantity of the exsudate, and its extension over wide ranges of organs and textures.

2. By the rapidity with which the effusion is brought about, where the stasis depends upon a pre-existent crasis.

3. By the often slight vascularity of the diseased texture; a circumstance due, it may be, to the blood-corpuscles not appearing prominently in the opaque, over vigorous plasma, or else to the exsudate, by its excessive quantity, soon leaving the blood-vessels exsanguine and collapsed.

4. By less adhesiveness,

5. By considerable fattiness of the exsudate.

The principal *metamorphosis* of the croupous exsudate is the aforesaid breaking down, and liquefying to a fluid more or less analogous to pus, which constitutes the so-called *liquid, purulent exsudate.*

This metamorphosis first of all affects the solidified blastema intervening between certain form-elements of the exsudate; only in an adherent portion of organizable fibrin does the latter undergo a change of texture—namely, to areolar tissue. When liquefied, it may be wholly reabsorbed, or may leave a residue in the shape of a fatty, curd-like, cretaceous pap, or, lastly, of a glutinous fluid, abounding in free fat and in salts of lime (in elementary granules, granulated cells, cholesterine crystals), which speedily thickens into a cretaceous concrement.

(*a*.) *Croupous Exsudation*, an abundant, proportionately to its contents in blood-globules or in blood-pigment more or less red (red hepatisation of lung), or grayish yellow mingling with green, opaque exsudate, consisting of a sod-like, fibro-laminated, or striated membranous basement, a large proportion of dotted substance, nucleated formations, dull granulated nuclei and nucleated cells. The nuclei are not influenced by acetic acid, beyond some little shrivelling, together with a sharpening, of their contours. This answers to the constitution of fibrin 3. It liquefies first in its basement mass, with change of texture of any adherent portion of organisable fibrin, to a pus-like fluid.

(*β*.) *Croupous Exsudation.* The above-mentioned characters, especially the opacity and the greenish coloration, are here more strongly developed. It consists, together with an amorphous blastema, of nucleus and cell formations, more or less akin to pus-nuclei and pus-cells, and of a predominating quantity of granulated substance. It adheres loosely to the exsudation surfaces and deliquesces rapidly. It answers to fibrin 4.

These exsudates occur most of all upon membranous expansions, upon mucous membranes (as the well-known croup), upon serous and synovial membranes, and in the substance of the lung. Again, they occur in areolar tissue, in the pia mater, on the convexity of the cerebral hemispheres, upon the endocardium and the internal blood-vessel membranes, in parenchymata, and upon the surface of both internal and external sores. Almost all the pneumoniæ, except those ending in slow resorption or induration of their product, belong to this class, and pre-eminently those in which the lung becomes enormously distended with a very copious, rapidly deliquescent yellow effusion, the stage of red hepatisation being in Hodgkin's opinion, here altogether wanting.

As the exsudates break down, they exert, especially after long-continued contact, the aforesaid corrosive, liquefying influence upon their substrata, occasioning ulcerous loss of substance, pulmonary abscess, destruction of serous membranes, ulcerous perforation of the thoracic, the abdominal, parietes, &c.

(γ.) *Croupous Exsudation.—Aphthous Exsudation,* a yellow, greenish-yellow, dingy-grayish, opaque product, wont, upon surfaces, to solidify into tough membranes, and then melt down, reducing the implicated textures to the same condition. This said product consists sometimes in simple destruction of the textures, sometimes in a blending into a variously discoloured, fetid, ichorous pulp, or into a tenacious, greasy slough, which tears like tinder.

These exsudates affect, with especial frequency, the mucous membranes, particularly those of the alimentary canal, of the urine-bladder, of the female sexual organs, with their follicles; secondly, external sores and ulcers; thirdly, the common integuments. Under this head belong thrush or aphthæ, diphtheritis, exudates upon the intestinal mucous membrane of the intestines, and of the colon in particular, as representing one form of dysentery, and of the uterus after child-birth;

corroding exudates upon external and internal wounded and ulcerated surfaces, for example, on the base of the typhous ulcer, white gangrene of the common integuments, hospital gangrene.

Croupous exsudation is occasionally the product of an intense inflammatory stasis, unconnected with any pre-existent crasis. It is, however, much more frequently the product of a stasis in which the croupous crasis has suddenly become localized.

(*c.*) A special form of fibrinous exsudation is the tuberculous—the true *fibrino-tuberculous*. It is a repetition both of simple, and, when engendered by inflammation, of croupous exsudation. It is characterised by a proneness to tarry long in its primitive, crude state, and eventually to soften down.

It exsudes almost, if not wholly, pure, or else blended with a certain proportion of organizable fibrin. In the former case it represents a homogeneous grey, or yellow, curd-like, brittle, fissured mass; upon serous membranes, a similar uniform layer of uneven stellate surface. In the second case, the tuberculous matter is imbedded in the shape of more or less crowded tubera clusters, or larger masses, within the other portion of the effusion, which has attained to various grades of textural development. In the last-mentioned case, more especially, tuberculous exsudation is, upon serous membranes, always characterised by clustering of the granules.

Tuberculo-croupous exsudation affects serous and mucous membranes, evincing a preference for those of the bronchial, of the alimentary, canal, of the uterus, and of the cavity of the tympanum. It invades the parenchyma of organs at all points, but most of all that of the lungs, as infiltrated tuberculous pneumonia of the lobar, or still more commonly the lobular form, the substance of lymphatic glands; lastly, the interior of follicles, and particularly the Peyerian capsules.

There are cases in which it may be but the local produce of a stasis. More commonly, however, this stasis is itself the localization of the tubercle crasis, and the tuberculous exsudate the product of general dyscrasial processes.

2. *Albuminous exsudation* occurs united to a proportion of fibrin as a fibrino-albuminous; or as a mere albuminous; or again, mingled with a certain proportion of serum, as a sero-albuminous exsudate.

Albuminous exsudates are marked by their fluidity, by their tenacious, ropy consistence, often by their abundance. They are colourless and resemble a thick synovia; or milk-white and opaque; or again blended with croupous fibrin and of a yellowish-white. Their turbidness, and also their thick porridge-like nature, are due to their quota of form-elements (elementary granules, nuclei, cells), to their proportion of fat, and to fatty conversion (granule-cells); or again, they are due to an act of coagulation, the albumen assuming, by virtue of a chemical conversion of the entire exsudate, the coagulate form. Coagula of this kind are for the most part soft, flocculent, forming, upon serous membranes, velvety deposits,—not unfrequently as the inner coating to a peripherous fibrinous coagulation.

The form-elements, present in various amount in the albuminous exsudate, are identical with those found in the fibrinous exsudate, nucleus and cell exhibiting themselves in multiform variety, ending with the perfect pus-cell.

It is very rare for albuminous exsudates to assume at once—that is, immediately on becoming effused, the solid form.

The changes which albuminous exsudates undergo, vary with the character of the albumen. In the one case, they enter into a progressive transformation of texture, which in fluid albumen obeys—in solidified albumen evades—the laws of the cell theory, thus determining hypertrophy of the areolar tissue, and induration and extinction of the parenchyma, for example, in the lungs. In dyscrasial constitution of the albumen, on the other hand, they have a decided tendency to liquefaction, to fatty conversion, whereby they become either fitted for resorption, or endowed with a corrosive power.

Their appearance is for the most part connected with a crasis, involving either a simple predominance of albumen (deficiency of fibrin) in the blood, or concurrently a dyscrasial condition of the former. To the first description belongs, for instance, the venous diathesis (Venosität), the concomitant of heart disease, of infancy, and of extreme age, of atrophy, of defibrination. The products are represented by colourless, adhesive, pasty exsudates, poor in form-elements, and, owing to the often low intensity of the stasis, largely diluted with serum;—sero-albuminous. To the second description belong the crases which attach to cutaneous affections, to typhus and the like, with their dull whitish effusion; and, again, the anomalous quality of albumen pertaining to the fibrino-croupous crasis, to pyæmia and the like, with their pus-like effusion. The stases are often of the asthenic or hypostatic kind, and run a protracted course.

3. *The serous, dropsical exsudate.*

Serous effusions are, generally speaking, either *merely serous* (blood-serum); or again from their containing a larger proportion of albumen, *albumino-serous*; or, lastly, owing to an admixture of fibrin, *fibrino-serous.* This gives rise to several important distinctions in their physical bearings. The mere serous effusion is a thin, watery, limpid, colourless, or pale yellowish, now and then reddish-yellow fluid, salt to the taste, and containing little, if any, albumen. A large proportion of albumen renders it tenacious, like a thin synovia. An admixture of fibrin manifests itself upon serous membranes as a peripherous coagulum of inconsiderable thickness, as a villous deposit, as a filamentous wide-meshed, network or finely membraned honeycomb, or as a flocculent cloud within the serous fluid. Or again, it may, in the shape of the so-called spurious fibrin, which solidifies tardily, perhaps only after cooling and coming in contact with the air, appear in the effused serum as soft, jelly-like, transparent, coagulate pellets, which are often found lodged within the aforesaid true fibrinous network, or honeycombed structure. Fibrino-serous effusion may be said to be invariably the product of an inflammatory stasis. It is simply fibrin-exsudate, with a notable preponderance of serum. It might well be denominated *fibrinated dropsy.*

Albumino-serous effusion is sometimes, like that previously described, the product of a not very intense, often of a protracted, stasis or simple congestion. As an example, may be cited the (inflammatory) œdema, encompassing the range of an inflammation. It occurs more commonly independent of the latter, *in the albumino-serous crasis,* as general œdema.

Mere serous effusion is the result of an excess of serum in the blood—the serous crasis; or else it is the product of acute and more frequently of chronic hyperæmia. In the latter case, the effusion seems to originate less from the capillaries than from the small thin-membraned veins. It represents genuine dropsy, and does not at all imply a foregone inflammatory stasis.

Serous effusion, as such, is not organisable. Albumen and fibrin, however, when blended with it in sufficient quantity, are susceptible of a somewhat tardy, structural change. It relaxes and tumefies the textures on becoming imbibed, destroys their contractility, and by long continued contact exerts, more especially upon the muscular fibres, a remarkable power-bereaving influence.

4. *Purulent and ichorous effusion.* These range immediately with fibrinous and albuminous exsudation, as also with their fibrino-serous, albumino-serous combinations. Purulent effusion seems, however, to be more especially akin to the fibrino-croupous exsudate.

The importance of these products renders it desirable to discuss them at greater length.

PUS, ICHOR.

No product of disease has, perhaps, been the subject of such zealous research as pus and ichor, and yet nowhere has a greater number of shortcomings been overlooked or glossed over than here. These we may be incompetent to remedy; we may, however, render some slight service to pathology if we can succeed in simply directing attention to them.

There are so many kinds of purulent-looking fluids, and there is so great an affinity of these fluids amongst each other, that, for the sake of discrimination, it is indispensable to establish one standard form of pus. Such a standard form is furnished in the pus of granulating, healing wounds, as well as in that of certain abscesses.

This *normal pus* is a homogeneous, cream-like, fatty, glutinous fluid, of a yellowish colour, of a flat, sweetish smell and taste, of a specific gravity of from 1030 to 1333 (Vogel), and when recent, of alkaline reaction.

It consists essentially of *pus-serum,* with certain form-elements, these being, besides molecular granules (elementary granules), the *pus-nucleus,* and the *pus-cell.* To these is to be added the pus-placenta, of which more hereafter.

The *pus-cell* is a spherical or oval—now smooth and even, transparent, thin membraned—now granulated and opaque, nucleated cell, which, under a magnifying power of 400 diameters, appears colourless, or faintly yellow, and measures from $\frac{1}{100}$ to $\frac{1}{80}$th of a millemeter in diameter.

Its granulated nucleus, firmly attached to the cell wall, is, in the translucent cell, visible without the aid of artificial expedients. In the granulated cell, on the contrary, it is rendered indistinct, if not totally obscured, by the contents of the cell, but is readily discernible on the application of acetic acid. It generally occupies from one half to two thirds of the cell's cavity, and in rare instances almost fills it up. Generally speaking, it is single; not unfrequently, however, it is manifestly composed of from two to five smaller corpuscles. Normal pus only rarely contains larger cells, with two, three, or four nuclei. Under the action of acetic acid, each pus-nucleus being brought out with sharper contours, as a spherical (according to Vogel, cupped) body, presents the well known characteristic phenomenon of indentation and eventual splitting. In other words, the nucleus, after passing through sundry modifications of shape, down to that of a trefoil, finally breaks up into two, three, or four sharply-defined corpuscles, no further soluble in acetic acid.

Besides the cell-inclosed nuclei there are present free nuclei. These are in like manner either single (perfected), or made up of from two to five corpuscles, and they exhibit the same phenomenon of indentation and splitting, when treated with acetic acid.

The molecular granules are present in various numbers, some scattered, others grouped together.

The contents of the cells are in some cases limpid, in others, owing to very minute granulations, nebulous. It is very common to find one compact group of pus-cells presenting every known gradation in the quality of their contents.

The development of the pus-cell is easily demonstrable, falsifying the assertion that the nuclei are artificially produced by chemical agency. For the most part from two to five of the larger molecules associate themselves into a group, and constitute, thus aggregated, an imperfect nucleus. By and by they coalesce, and present a simple, finished nucleus—a fabric reducible, by the agency of acetic acid, to the very same elements.

The nucleus now becomes surrounded, often immediately, with a cell wall, so closely fitted at first, as to require the endosmotic agency of water, or dilute acetic acid to disconnect it, and render it cognizable. Many nuclei, however, become previously endued with a delicate nebulous deposit, which by and by puts on a circumscribing cell-envelope, and assumes the contour of the cell.

These formations are, to a greater or smaller extent, always discernible in genuine pus, in the progress of germination.

Cells devoid of nuclei,—clear, transparent cells, which have to create a nucleus out of their own materials, are rare.

The phenomena of endosmosis and exosmosis bring out the pus globules with great clearness.

The chemical relations of the pus-cell are not without their weight in reference to its constitution, and to its recognition.

Dilute acids, for example, dilute hydrochloric, oxalic, tartaric, but especially acetic acids, have the effect of tumefying, loosening, attenuating, bursting, without entirely dissolving the pus-cells, whilst upon the nuclei they produce the above-mentioned appearance, first, of greater distinctness of outline, then of indentation, and lastly, of disruption.

Caustic alkalies and their carbonates convert the pus-cells into a jelly-like, granulated substance.

Thin solutions of certain saline substances, as for instance, of chloride of sodium, hydrochlorate of ammonia, nitrate of potash, iodide of potassium, with many others, cause first the disappearance of the sheaths, and secondly, the swelling up of the nuclei into a shapeless grume.

A solution of borax acts like the alkalies, only less rapidly. Metallic salts, alcohol, tannic acid, &c., which coagulate fluid albumen, render the pus-globules shrunken, nebulous, and opaque.

In the blood, in urine, in mucus, and in saliva, the pus-cells are preserved unchanged; bile, on the contrary, occasions a disappearance of the sheaths, and a bloated aspect of the nuclei.

From these facts, and from further experiments in the same direction, Lehmann and Messerchmidt draw the following conclusions:

1. The sheath of pus-cells, turgescent in acids, soluble in solutions of caustic alkalies, and of their saline conjunctions, is identical with a protein-compound which may be artificially

produced out of albumen, deposited by water, and redissolved by alkaline salts, and acetic acid—a modified albumen, poor in salts, constituting a transition stage to fibrin—fibrin *a*.

2. The nucleus, insoluble in acetic acid, soluble in solutions of alkalies, turgesent in solutions of salts, a protein-compound similar to the venous fibrin, turgescent in salines—fibrin *b*.

3. The third substance, namely, the molecules accompanying the pus-cells, forming part of the contents of the opaque granulated cells, and even exhibited in the nuclei (the nucleus-corpuscles of Lehmann and Messerschmidt), are uninfluenced by alkalies or borax, and are regarded by Lehmann and Messerchmidt, as a substance analogous to the essential constituent of horny texture. They, however, partly consist, as Vogel rightly maintains, of fat.

Besides these elements, pus not unfrequently contains cholesterine-crystals, crystals of ammonio-phosphate of magnesia, animalcules, &c.

Like Henle, we have been unable to satisfy ourselves of the above-mentioned effect of alkaline salts or of borax solution. In the changes wrought by the application of thin solutions, we recognise the phenomena of endosmosis down to rupture of the sheath of the pus-cell; in the changes wrought by the application of saturated solutions, a shrivelling thereof.

The pus-serum in which, when at rest, the pus-cells gravitate, has the composition of blood-serum, with some difference, however, in the relative proportions of its constituents; fat, for example, predominating. With respect to pyin, to which we shall afterwards have to recur, our belief is that it is not a constituent of normal pus at all.

Pure pus we believe to be an albuminous exsudate, out of which, like other elementary bodies, the pus-cell becomes developed by virtue of a specific conversion. This process, the so-called development of pus out of fluid blastema, occurs upon surfaces—upon mucous membranes, upon the external skin, upon open wounds, in abscesses. This pus exsudate, however, enters frequently into combination with fibrinous exsudates of different kinds.

Of these, we could first mention the combination with the fibrino-croupous exsudate, because this furnishes the base of the so-called development of pus out of solid blastema. Whether, or how far, we participate in this view, will appear in the sequel.

Both upon surfaces and within parenchymata, a solidifying fibrinous blastema is frequently thrown out. A careful inspection will show, imbedded in the solid basement, as also floating in the sero-albuminous fluid, molecular granules and genuine pus-nuclei and pus-cells. Together with these are always found a few nucleus and cell-forms, which vary in their relations, more especially to acetic acid. The solid basement manifests itself as croupous fibrin, which liquefies, incorporates the aforesaid elements, and is distinguished by its great abundance of the most minute molecules. This combination of pus with fibrino-croupous exsudate, constitutes the so-called solid pus or the pus-plug, and is that upon which the breaking down, the softening, of inflammatory induration, abscess, and the like depend. The pus-cell is always developed out of the sero-albuminous moisture pertaining to the fibrin-exsudate, never out of or at the expense of the latter itself, the liquefaction of which implies a metamorphosis. This pus, in virtue of its constituent, croupous fibrin, contains pyin.

Another combination of the pus-exsudate, is that with plastic fibrin exsudate. This frequently, but not invariably, furnishes in pus the basis of new solid textures, of regeneration, of the cicatrix. The fibrin-exsudate determines the pus-placenta.

Pus may, without impairment of its primitive character, present various anomalies, for example:

(*a.*) Watery pus; the pus-cells having become bloated in the preternaturally thin medium.

(*b.*) Preternaturally saturated pus-serum, as containing more saline and albuminous matter. The pus-cells appear less turgescent, smaller, shrivelled, denticulated.

(*c.*) Pus which has become acid exhibits the nuclei more distinctly, more sharply defined, and, it may be, in a slight measure ruptured, within a transparent membranous sheath.

(*d.*) Various admixtures, as blood, mucus, epithelial, and other textural débris. It is to be remarked, however, that, by certain admixtures, for instance, of fæcal matter, of decomposed urine, and, again, by its acid conversion, pus may become transformed into a corrosive fluid, and its secreting texture goaded into the production of ichor.

The nearest approach to pus is found in broken down fibrino-croupous exsudates. It has been seen that these frequently enter into a combination with true pus-effusion, and the liquefaction of the fibrino-croupous elements in pus-effusion constitutes what is termed the development of pus out of a solid blastema. Broken down, pus-like, fibrino-croupous exsudate is always marked by its fluid parts holding in suspension a large proportion of the most delicate nebulous molecules, and is distinguished from pus by the relations of the coexistent nuclei and cells. These exsudation elements, namely, manifest, as we have stated, on the one hand, an insensibility towards acetic acid, under the influence of which, by an evident condensation and shrinking, the cell-walls and contours of the nuclei are brought more distinctly into relief; or it may be that the cell-walls and cell-contents are rendered clearer, whilst the nucleus becomes condensed and more sharply defined.

On the other hand, these cells approximate to the character of the pus-cell, the nucleus exhibiting, with the disappearance of the cell-wall, to a various extent the phenomena of denticulation and splitting. It is, in our opinion, the broken down fibrino-croupous exsudate, either alone or blended with true pus, that constitutes the pyin-holding pus form. It has been stated that these pus-like exsudates frequently manifest a corrosive, deliquescent influence upon the textures. Not being organizable, they are extremely prone to further decomposition, and to assume the nature of ichor. They furnish forth the majority of cases of internal suppuration, of constitutional pus deposits, of abscesses.

Ichor, which, in broken down croupous exsudates, often closely resembles pus in appearance, is distinguished from the bland nature of true pus, by its corroding influence upon the textures, and upon the form-elements developed out of its protein substances. It is only under such a state of things that a fluid can be recognised as ichor. That met with after death varies infinitely. A chemical examination of it embodying what is essential, and simplifying what seems differential, is still wanting. Its degree of corrosiveness varies equally. Ichorous exsudates are now thin, serous fluids; now albuminous, viscid, limpid or flocculent, emulsive and fatty, thickish, colourless, or yellowish, yellowish green, puriform, whitish, creamy. Or, owing to the presence of blood-corpuscles and of blood-pigment, they are of variously shaded red, dingy brown, greenish brown, chocolate coloured. Again they are ammoniacal, hydro-sulphuretted, rancid or sour smelling, acid or alkaline, and apt to produce upon the skin of the dissector a tingling or smarting sensation. These fluids, minutely examined, are found to contain variously sized elementary granules, down to the finest molecular mass, nuclei and cells, of the character of exsudation- and pus-cells, partly stunted in their development, partly owing to the saline, alkaline, or acid condition of the ichor, shrivelled, jagged, lax, diffluent, the pus-nuclei being in the act of denticulation and splitting.

They further contain fibrinous coagula of various kinds, in different grades of spontaneous reduction into pulpy masses, coagula out of casein and pyin substances.

Finally, they yield crystalline salts and textural débris in the act of breaking down, blood-corpuscles, animalcules, &c.

Even ichor enters into combination with fibrinous exsudates, especially the fibrino-croupous; and, just as in the case of pus, there is, besides the fluid product ichor, another ichor developed out of consolidated blastema. Having now described both pus and ichor, this appears to us a proper place for the establishment of certain marks necessary for a due discrimination between the two.

The bland properties of normal pus are acknowledged; but how does this characteristic tally with the manifest destruction of tissues implicated in the formation of pus? The following remarks may tend to throw some light upon this point:

(*a.*) The destruction attendant upon pus-exsudation is limited to necrosis of the textural elements involved. But this necrosis is due to the intercepted supply of blood and to forcible disjunction; not to that chemical corrosion and that resolution of the textures which result from ichorous discharge.

(*b.*) That the bland nature of pus is so frequently questioned, arises from products being so often regarded as pus, which are not so in reality, and which either originally possessed, or have acquired the property of corrosiveness. Such products very commonly form the contents of shut abscesses. Normal pus very often acquires a corroding property through long seclusion within abscesses, which, when opened, forthwith secrete a normal, bland pus.

One of the most remarkable phenomena in the process of suppuration, is the formation of *flesh granulations*. These granulations present, with reference to their character and further development, two marked distinctions:

(*a*.) They consist, together with a small proportion of intercellular or bond substance, of primary cells. These cells emerge, together with the pus-cells, from a common albuminous blastema, and out of them are called forth, conformably with the laws of the cell theory, those elementary fibrils which ultimately compose the cicatrix.

(*b*.) They consist of a fibrinous blastema which, exsuding conjointly with the pus, solidifies upon the suppurating surface, and yields, by immediate splitting, the fibrous texture of the cicatrix. Into both these scar-bases enters a new generation of blood-vessels, answering to those of abiding suppuration. They determine the healing of wounds by the second intention.

The formation of flesh-granules is not decisive evidence of a bland, benignant pus. It may accompany, and even luxuriate under, the production of ichor. In the one case, however, the granulations are marked by their durability, and by their further textural development, whilst in the other, they form but to be corroded and redissolved by contact with the ichorous fluid.

Flesh-granulations accompanying the production of pus in or upon heterologous growths, have, for the most part, the significance of textural elements of such growths.

The tendency of pus (and of ichor) to vent itself externally, is commonly overrated. Pus deposits are often deeply encysted within organs, and a passage to without often needs to be made artificially, in order to prevent the fluid from burrowing.

The assertion that extensive exsudates are especially liable to become converted into pus, is ill founded, if magnitude of the exsudate be the assumed condition of such conversion. Doubtless what led to the opinion, was the liquefaction of the commonly very massive croupous exsudates, together with the circumstance that pus-blastema, either pure or combined with croupous fibrin, very often becomes effused in great quantity. Apart, however, from quantity, the puriform quality of an exsudate is invariably due to inflammatory stasis. This alone, and not the quantity, can determine the development of the elements of pus.

Purulent and ichorous exsudates are met with not alone in the localities assigned to normal pus. They often occur in great quantity upon serous and synovial membranes, and in areolar tissue, more especially the subcutaneous and submucous, as also in certain of the more lax, deep-seated collections of this tissue, for example, in the mediastina, in the posterior circumference of the cæcum, around the rectum, and the like. Lastly, they take place upon mucous membranes, within soft parenchymata, and in bone.

The following are the metamorphoses which the said exsudates, if not excreted, undergo;

(*a.*) *Transformation of texture*, appearing, after the process above described, in the form of so-called granulations.

(*b.*) *Dissolution* affects both the form-elements (cells, nuclei), and the fluid intercellular substance, in the shape of various unknown chemical transformations. Under this head may be brought the septic decompositions, suffered by these exsudates under peculiar circumstances; for instance, through long stagnation, through contact with the atmosphere, through the effect of medicinal substances, and the like. These may cause the degradation of bland pus into ichor, and of ichor a step lower in the scale.

(*c.*) *Fatty conversion* in the formative process of granule cells, frequently combined with the simultaneous deposition of the salts of lime, (cretefaction).

(*d.*) *Resorption.*

The manifold ways in which pus substantively, or as pus serum, enters the circulation, and there occasions pyæmia, cannot all concern us here. They were before adverted to, and will be further and more amply noticed under the head of pyæmia.

Here we have to consider, the resorption of pus in a more restricted sense, and irrespectively of pyæmia.

This resorption can apply only to the serum or plasma of pus. To pus or ichor in their totality, that is to say, their form-elements included, it can only apply after they have undergone liquefaction or fatty conversion. Upon the mode of liquefaction depends, in the case of pus, the nature of the consecutive phenomena. If it consist in a putrid decomposition, the noxious effects of septic poisoning of the blood may ensue.

Pus may either become completely reabsorbed, or leave within its former nidus a residue of fat in the shape of discrete or aggregate fat molecules and cholesterine crystals, within a glutinous fluid, or in the shape of chalk incrustation and concretion.

The depôt of pus and of ichor (abscess) heals, through the subsidence of pus and ichor production, and through the organizable products of inflammation existing in the walls of the abscess, with new supplementary ones, undergoing transformation into vascularised textures. Pending this act, the contents of the depôt are in the manner before specified, entirely or partially reabsorbed. According to the measure of resorption, the walls of the abscess shrink together and ultimately coalesce into a solid cicatrix, or else include an inspissated fatty or chalky, or lardo-cretaceous residue of the primitive contents.

Pus and ichor are the product, at one time of a mere local inflammatory stasis, at another time of a localized pre-existent crasis, namely, general pyæmia. In the latter case, the production is characterised by the rapidity of its occurrence.

Suppuration in open abscesses and upon granulating sores is peculiarly chronic in its course. This process manifests itself as a protracted stasis, communicated from the original textures to the embryonic new-growths which the granulations with their newly acquired vascular apparatus have given rise to. Taken in another point of view, the granulations appear to stand to pus-formation in a relation which earlier pathologists signified by the terms, *pyogenous membrane*, a pus-secreting apparatus. Looking at the often slight intensity of the inflammatory symptoms, the analogy between a granulating wound (abscess) and a secreting organ,—between pus and a secretion prepared by those elementary bodies, cells, appears so great, and the current comparison with a mucous membrane and its product so apt, as forcibly to recur to us, even at the present day. Thus, no sooner have the growths which presided over the secretion—in other words, the granulations—become exalted into textures, than the secretion itself fails.

Referring certain particulars connected with the anatomical doctrine of exsudates to the head of crases, we have still to consider, as kindred with ichorous exsudates, *diffluent exsudates*, and with them *hemorrhagic effusion*.

(*a.*) *Solvent exsudates.* Akin to ichorous exsudates, they are marked by their destructiveness to subjacent textures, by the obviously solvent character of their effects, and by the absence of any outward tokens to denote their mischievous character.

These are products which, owing to the corrosion of textures accompanying the very act of their exsudation, are rarely to be met with in their simplicity, products which display immense variety in their physical properties. As the extreme limits of a long series, we find, on the one side, a coagulable, fibrinous exsudate which has the effect of slightly corroding the subjacent membranous substance. On the other side we have a thin exsudate, variously discoloured, which reduces the textures, extensively, to a dingy brown, chocolate coloured, inky (hemorrhagic), or greenish, pulpy, tinderlike, fetid, slough. This last-mentioned exsudate represents the processes which Boër described in the uterus as putrescence, a term quite deserving of application to the same condition in other parts. Midway between these two extremes, we encounter the most remarkable—however seemingly insignificant—thin, serous, sero-albuminous, tenacious, paste-like, sero-purulent, almost colourless, or again yellowish, reddish-yellow, exsudates, in contact with which the textures are resolved according to their degree of injection, into a pale, or into a more or less deeply-reddened pulp.

Their seat, always diffuse, is most particularly the mucous membrane of the intestinal tract, and most commonly of the colon, not rarely of their follicles (in the shape of diarrhœa or dysentery), and, lastly, of the uterus, as puerperal affections following childbirth. Of the principle upon which this liquefiant destruction of the tissues depends, nothing is known beyond its frequent acid reaction, nor has any crasis corresponding to it been recognised.

(*b.*) The *hemorrhagic exsudate.*

It is indispensable in the first place to discriminate between exsudates reddened by blood pigment only, and those which contain substantive blood, that is, blood-corpuscles.

The former are met with in all dyscrases, both acute and chronic, in which, owing to defibrination, to decomposition of the fibrin, or to diminished proportion of salts, blood-pigment is transferred from the blood-corpuscles to the blood-serum. Thus, exsudates occurring during the progress of scurvy, of typhus, of gangrene, of the drunkard's dyscrasis, of putrid exanthemata, are stained with adherent blood-pigment. Those containing blood in its totality, and the red colour of which results from blood-corpuscles, are the true hemorrhagic exsudates.

Holding fast this distinction, we shall be enabled partly to infer from physiological reasoning, partly to prove by the exact method, the origin and import of hemorrhagic effusion.

We have seen that, in every inflammation, at the stage of congestion and stasis, there occur extravasations of blood, proportionate in extent to the vascularity of the organ, to the magnitude of its congestion and stasis, and lastly, to the laxity and vulnerability of its texture. That this bleeding takes place out of lacerated, or in some wise opened, vessels by extravasation, and not by transudation, is evident from the absence in the walls of blood-vessels of pores equal to the transudation of blood-corpuscles. This is the rule with respect to hemorrhage in textures like those of the brain or the lungs. The difficulty is, and always has been, to explain hemorrhagic effusion occurring upon serous membranes,—a formation so given to effusion in no way hemorrhagic. Upon this point it is to be observed:

(*a.*) A primitive genuine hemorrhagic exsudate (not merely blood-stained), if it really ever occur upon serous membranes, occurs only as a rare exception.

(*b.*) Hemorrhagic exsudates upon serous membranes are, almost without exception, the result of hemorrhage from the blood-vessels of a spurious membrane in the act of becoming organized; in other words, from the product of a previous inflammation of the serous tunic. This hemorrhage may be an independent act, or it may be the concomitant of inflammation propagated from the serous coat to its pseudo-membranous duplicate. This is, in fact, usually the case. Such exsudates are secondary ones.

The facility with which hemorrhage takes place from these new-growths, is explicable on the ground of their imperfect organization, both as regards change of texture and the development of blood-vessels. An inflammation early set up in such a new formation, encounters a lax, soft, lacerable growth, involving an incomplete, soft, and delicately membraned vascular apparatus, with anastomoses as yet unclosed; blood-vessels which, when urged into congestion and stasis, readily give way, or possibly force a passage from their free and as yet unanastomosed ends into the substance, and through this into the cavity, of the serous membrane.

Accordingly, under these stereotype conditions, hemorrhagic exsudation is precisely what extravasation is during the course of an inflammation in the laxer textures, namely, *exsudation plus hemorrhage.*

Hemorrhage being, however, the consequence not alone of mechanical laceration, but also of a softening or a corrosion of the vessels, it is intelligible how ichorous exsudates, and the solvent exsudates generally, may put on a hemorrhagic character.

Hemorrhagic exsudation seems to stand in an especial relation to tuberculosis, and is dreaded chiefly because the latter is assumed to be its source. It is important to be clear upon this point:

(*a.*) It is quite true that it is very frequently a partially tuberculized new growth (pseudo-membrane) in which the hemorrhagic process occurs. Still there are very notable exceptions to this.

(*b.*) Tuberculized growths appear to be peculiarly liable to inflammation; tubercle being wont to set up reactive processes of inflammation everywhere in its circumference.

In this way the hemorrhagic inflammatory process often concurs with local tuberculosis, without directly depending upon the tuberculous crasis. That tuberculosis acts as the source of hemorrhagic effusion is rendered probable by experience, but it is by no means proved.

In like manner, hemorrhage and hemorrhagic exsudation break forth in the midst of carcinomatous growths, or in pseudo-strata, of the same character, upon mucous membranes or within serous sacs.

In quantity, the extravasate mingling with the exsudate varies considerably, and it may be either intimately blended both with the coagula and with the fluid portion of the exsudate, or separated from it in the shape of pellet-like clots.

Apart from the aforesaid conditions of its appearance, and of its relation to tuberculosis, it is of evil omen only in proportion to the loss of blood entailed by it, and to the previously reduced vital strength of the patient.

The hemorrhagic exsudate is not organizable, or only very tardily so. This refers more particularly to the exsudate; the blood-corpuscles after long remaining unaltered at length become dissolved, leaving their pigment to undergo the changes elsewhere described. The exsudate-portion, answering to the character of its base, very commonly retains its rudimental condition, which in the majority of cases is tuberculous.

It would seem advantageous, before concluding, to revert to a few points already touched upon, relative to the habitudes of exsudates in and upon diseased structures.

Exsudates are deposited more or less uniformly between the elementary parts of textures. This is contingent upon the more or less uniform density and cohesion of the textures, as also upon the number of blood-vessels present, and their mode of distribution; for example, the striated exsudates, following the linear arrangement of the blood-vessels in tendons and ligaments.

When copious effusion takes place suddenly and violently in the laxer structures, for example, in the brain, the exsudate becomes established through the forcible separation and laceration of the natural textures.

In the inflammation of membranes the exsudate is, as we have stated, for the most part thrown out upon the free surface. In the inflammation of glands similar effusion takes place into their respective cavities—the uriniferous tubules, Malpighian bodies, and the like.

Coagulable exsudates solidify upon the surface of inflamed membranes, and are commonly termed spurious membranes. Upon serous membranes they occasion agglutination of the serous surfaces.

ISSUES OF INFLAMMATION.

The so-called issues of inflammation comprehend a variety of processes. They concern either the inflammation itself or its products and the involved structures; that is to say, they embrace, in the latter relation, the changes which both the inflammatory products and the textures themselves undergo.

To the former category belong:

1. *Resolution or dispersion of the inflammation.*

To the second belong:

2. *Reliquefaction and resorption* of the inflammatory products.

3. *Abiding of the inflammatory products in various forms,* including, amongst others, the issue in induration, and in inflammatory hypertrophy.

4. *Suppuration,* ichorous, ulcerous destruction of textures.

We cannot ourselves regard suppuration as an issue of inflammation. Wherefore we have treated of pus and ichor as of products of inflammation, under the head of "exsudates."

Of the issue of inflammation in gangrene, of so-called inflammatory gangrene, we cannot well treat separately. We therefore refer the consideration of this point to a subsequent chapter, to be devoted to the subject of gangrene.

1. *Resolution of the Inflammation.*—Issue in *resolution* relates directly to the inflammatory process. It consists in a cessation of the latter, previously to any act of effusion; that is, in a reduction of the existing stasis.

As determining conditions we may adduce:

(*a.*) Cessation of the efficient cause of inflammation; and, as a consequence, cessation of the palsy of the blood-vessels, and returning contractility of the latter.

(*b.*) Reinforced impulse from the arteries, brought about by the said contractility of the capillaries, one effect of which is a return of the phenomenon of oscillating motion in the arrested blood-column.

(*c.*) Liberation of the blood-corpuscles from a state of mutual cohesion, through endosmosis of the exsuded blood-serum into the vessels charged with concentrated plasma. The blood-corpuscles lose the flattened condition and deep colour which they had acquired in the stasis, swell out, and become spherical and at the same time paler.

(*d.*) The uninterrupted circulation in the capillaries surrounding an inflammation, plays its part likewise, portions of the stagnant blood-column being (according to Emmert) forcibly separated by the laving current, and a passage forced here and there through an entire capillary range.

Obstacles sometimes present themselves to the resolution of inflammation; and even when the process is accomplished, certain residua are left behind:

(1.) The blood-corpuscles are so firmly wedged in the dilated capillaries, as to resist both the contraction of the latter and the increased impulse from the arteries. This occasions a protracted stasis of a mechanical nature.

(2.) Even after removal of the stasis, a certain degree of palsy and dilatation of the capillaries may remain entailed. The part previously inflamed continues in a state of hyperæmia, and prone to relapse into inflammation. This tendency increases greatly upon repetition. The resolution of local inflammations dependent on external causes, is common only where these causes are slight.

Inflammations dependent upon internal dyscrasial influences rarely terminate thus. An essential condition of their so doing is the extinction of the dyscrasis. If without this the inflammation take such an issue, it will localize itself in other organs standing in the relation of sympathy with that originally affected.

Even this favorable consummation of the inflammatory act is not a matter of indifference for the organism, seeing that plasma, altered by the previous stasis, is copiously received back again into the blood. The consequences are obvious, and directly commensurate with the extent of the imflammation.

2. *Re-liquefaction and resorption of the inflammatory products.*—This issue of inflammation is contingent upon previous exsudation, and consists in resorption of the products. It takes place with greater or less facility, according to the measure of the exsudation, and to its degree of solidification. It succeeds either completely or incompletely, and hereupon it depends,

whether in the sequel the diseased organ recovers its normal condition altogether, or only partially and imperfectly.

Fluid exsudates are naturally susceptible of resorption.

Solid exsudates become adapted for resorption by preliminary solution,—corrosion through blood-serum,—or else by disintegration, with various changes in their chemical composition.

Elementary bodies must be previously dissolved, in order to become adapted for resorption, which process takes place both through the blood-vessels and the lymphatics.

The consequences of resorption differ with the primitive quality of the exsudate, with the mode of its preliminary solution and of its chemical transformation, and with the quantity reabsorbed. Finally they differ accordingly as the resorption takes place chiefly through the lymphatics or directly into the sanguineous current.

In this issue of inflammation is comprehended *wasting of the textures through inflammation.* It consists in the elements of the textures being themselves liable to become reabsorbed along with the products of inflammation. This is owing to the textural elements, within the range of inflammation, becoming functionally disabled by mechanical pressure, to the impediment to their nutrition offered by the effusion; the result being the dissolution and resorption of those elements. This termination is especially frequent in delicate, vulnerable textures, in very copious effusion, and where the latter, being solidified, is susceptible only of very tardy resorption. In this manner is the substance of the brain, of muscle, of kidney, and the like, destroyed within the range of inflammation, its place becoming occupied by a cavity, or by multilocular cavities bounded by scar texture. Where these are small and numerous, they beget a loosening, a rarefaction of the textures, as, for example, in the condition termed cell-infiltration in the brain. In hollow structures, for example the Graafian vesicles, the contents, altered by the exsudate and its metamorphoses, are absorbed, and the organ becomes extinct.

8. *Abiding of the inflammatory product.*—The products of inflammation are retained bodily, or after imperfect resorption, partially, in their original, or it may be in an altered shape and constitution. Conformably with what has been stated, the exsudates continue—

1. In their original crude state, as entirely amorphous masses, or more commonly in a condition bordering upon this, of incipient textural development, in the shape of molecular granules, of nucleus and cell-formation, or in the case of consolidated blastemata, in the laminated and fibrous structure engendered by the coagulative process itself.

2. Or they break up sooner or later, and abide in a state of final cretaceous or fatty conversion.

3. Solid exsudates waste away, and condense and shrivel into a horny substance.

4. They become organized, attaining thus to various grades of development. In fluid exsudates this development follows the laws of the cell theory, whilst, in the organizable, solid, fibrin-exsudates it consists in immediate fibrillation through dissilience.

The exsudates enter, bodily, into a uniform textural change. This change may, however, conformably with the frequent primitive impurity of blastemata, be in many cases unequable. One portion of the exsudate may attain a higher gradation and represent permanent textures, whilst another may be arrested at an embryonic stage, and there liquefy and become reabsorbed, or, like pus and ichor, qualify itself essentially for excretion.

A textural conversion of exsudates may be of a nature very similar to, if not identical with, the normal structure, in its anatomical, chemical, and functional relations. Or, again, it may involve one or more heterologous formations, pus and cancer, for instance.

It is more particularly areolar tissue, and the various fibroid textures tending to the final composition of the latter; cartilage in the process of ossification; and bone with its penetrating vessels, that are here referred to as organized products of inflammation.

These often serve to compensate for lost parts, as also for the filling up of vacant spaces wrought by the retraction of normal textures after injuries, regeneration in muscles for instance.

The regeneration is complete; or it is imperfect, being accomplished by means of a texture not homologous with the lost one,—as in the cicatrix. This, again, may be permanent, or it may be provisional only, and about to disappear after becoming endowed with textural elements identical with the normal ones,—the nerve-cicatrix, for example.

Where permanent exsudates do not serve for compensation, they occasion an increase of mass in the diseased organ. They are then—

(*a.*) Uniformly interposed between the elements of the normal texture. This determines an inflammatory hypertrophy, which, perhaps, occurs in a genuine form only in areolar tissue and in bone.

(*b.*) Or they form in a larger circumscribed mass, distinct from the normal texture,—a tumor. These local collections of cicatrix texture occur in muscle (the heart) and in all parenchymata. Upon serous membranes, they constitute the various organized pseudo-membranes and membranaceous adhesions. We have here further to remark:

1. In bulky, and particularly in solid, wasting exsudates, or such as are in progress of transformation into dense, shrivelling (fibroid) structures, any textural elements which they have embraced become atrophied by pressure and tension, hindrance to their nutrition, and the suppression of their function. Even contiguous textures waste away, owing to arrested or impaired function—for instance, the muscular apparatus of respiration over thick, resistent, shrivelling, pleuritic effusion.

2. In hollow organs the abiding of exsudates not rarely occasions a hypertrophic development to cystiform dilatations, with transformation of the texture of their walls and contents. As examples, we may refer to the degradation of glandular acini, and of the follicles into cysts. (See Cyst.)

3. Solid exsudates determine, through increased consistency and density of the textures previously inflamed, the issue of the inflammation in so termed *induration*. On the other hand, the abiding of soft liquid exsudates results in relaxation, softening, lacerability of the textures.

4. Exsudates are often found to linger under several combined forms, with which, moreover, both resorption and suppuration may have concurred.

4. *Ulceration, Ichorous destruction.*—It consists in a wasting of the textures from the corrosive quality of the exsudate. Herein ulcerous consumption of the textures differs from the loss of substance which inflamed textures undergo,

within the best conditioned exudates, through necrosis and absorption.

To be productive of such a wasting process, the exsudate must needs be fluid, whether originally so, or liquefied out of a solid blastema. Its corrosive influence upon the textures is sufficient to confirm its character as genuine ichor.

The mode in which textures in contact with ichor become destroyed, varies with the principle upon which its corrosive nature depends. The exulceration takes sometimes an acute, sometimes a chronic course. Large textural masses are not rarely destroyed within a short space of time. The destruction is marked, now by superficial extension, now by a burrowing propensity. In the former instance, it depends frequently upon a special relation of the inflammatory process to superficial textural expansion. In the other case, some heterologous formation reproduced again and again, at the base of the ulcer, upholds the inflammation, and with it the ichorous discharge.

In the chronic form, the ulcer, like the pus membrane, simulates, in the production of flesh-granules, a natural process of secretion.

All textures are not equally prone to ulcerative destruction. Under like circumstances, tender, young, budding growths are the most readily destroyed.

GANGRENE, NECROSIS.

Under *gangrene, necrosis,* is understood the death of an organ or part, as manifested by the more or less rapid breaking down and chemical decomposition of its texture. Gangrene may affect both soft and solid structures, the bones, for instance, or even fluids, as in necrosis or sepsis of the blood. The breaking down of solid structures, is generally a slow process, whilst in soft, juicy textures, and in fluids, it is rapidly consummated. Like normal textures, new formations of every kind,—tumors, exsudates, pus,—are liable to become necrosed. Fluids degenerate through necrosis to *gangrenous ichor,* the most infectious and destructive of its tribe.

A general characteristic of gangrene is not easily given, so manifold are its forms, and so various its causes. Soft parenchymata commonly break down to a diffluent pulp, marked by a high degree of discoloration and of fetor. Exceptions are, however, numerously afforded in gangrene of the bones, mummifying, white gangrene.

Gangrene has the import sometimes of a local, sometimes of a symptom of general, disease. The conditions necessary to the former case are nearly reducible to arrested afflux of blood, that is, stasis. It may begin by attacking fluid parts, and especially the blood, and extend from these to solid structures, or it may affect them all at once.

Gangrene is developed—

1. Out of *absolute blood-stasis*, which may occur under various circumstances:

(*a.*) Every hyperæmia in organs, or sections of organs, paralysed or enfeebled, or obnoxious to debilitating influences, may degenerate into absolute stasis. This applies particularly to asthenic, hypostatic hyperæmia in torpid peripherous organs, vegetating, so to say, imperfectly under the embarrassment of continued pressure.

(*b.*) Mechanical hyperæmia frequently becomes absolute stasis, as observed in incarcerated, strangulated organs, and as a consequence of extensive plugging of the returning vessels in the lower extremities.

(*c.*) Every inflammatory stasis may degenerate into absolute stasis, more particularly those hypostatic and asthenic inflammations which occur in organs already diseased, paralysed, or depressed by violent external influences, such as concussion, contusion, cold, or heat. An inflammation consequent upon influences directly or indirectly debilitating, may acquire, during its progress, a tendency to absolute stasis.

In *absolute* stasis, the blood undergoes gangrenous decomposition. Hence the blood is the portion originally necrosed and dissolved. It exsudes in a state of gangrenous decomposition, and in the form of ichor, through the walls of blood-vessels, engendering the same gangrenous decomposition both in these and in the surrounding textures. This event gives rise to the most ordinary and most developed form of moist gangrene, in which the textures are, through the medium of the blood, broken down to a dark-coloured, friable and lacerable, diffluent, and highly fetid pulp. The dark discoloration, however, of gangrene thus developed, is subject to various modifications due to certain elementary products, which the inflammatory stasis has generated both within the blood-vessels and without.

The progress of this gangrene is more or less acute, the gangrenous dissolution of tissues, already referred to under the term putrescence, being particularly marked by the rapidity of its course.

2. *Gangrene is determined by failure in the supply of blood:*

(*a.*) In impermeability of large arteries,—high degree of coarctation, and complete obstruction—consequent upon arteritis and ossification.

Here the gangrene, for the most part, takes the form of comparatively dry, black, mummifying gangrene.

(*b.*) As the result of the immediate compression and tension of a part; for instance, in incarcerated hernia.

(*c.*) As a consequence of the local destruction of blood-vessels, the denudation of parts of attaching and blood-supplying textures,—bones, for example, of their external and internal periosteum; the common integuments, of their supporting areolar tissue; the peritoneum, of its subjacent layers; isolation of the pulmonary pleura over cavities of the lung.

The gangrene appears as a white or yellowish white slough.

To this category belongs the necrosis of smaller textural parts, loosened mechanically by exsudation or by ulceration.

(*d.*) Extensive impermeability of the capillaries and minute vessels when plugged with coagula, or compressed by surrounding exsudates.

In the last mentioned case, the gangrene is dependent upon inflammation. To this kind of gangrene, textures poor in blood-vessels, such as compact bones, callosities, &c., are especially obnoxious. The colour of the necrosed textures differs with the different nature of the coagulation, and of its exsudate. Answering to the croupous character of bulky exsudates, the textures involved in the necrosis commonly assume a yellow or yellowish-green hue.

3. *The gangrene is the expression, the localization of an anomaly in the blood-crase,* either directly ingrafted by infection (contagion), or developed out of other crases; a putrid decomposition of the circulating fluid. Blood so poisoned, especially if brought into stasis or into coagulation, possesses, in common

with the exsudates thrown out by it, an inherent tendency to gangrenous dissolution.

It has been already stated, that several varieties of gangrene are recognised:.

1. *Gangrene* developed out of an internal cause is distinguished, by the designation of *primary gangrene,* from that arising out of a predominant external cause.

2. *Hot, acute, inflammatory gangrene.—True gangrene.* In what wise inflammation leads to gangrene, is sufficiently clear from the foregoing.

(*a.*) The inflammatory stasis, owing to its very intensity, to pre-existent debility of the diseased textures, or, lastly, to weakening influences exercised during its progress, degenerates into absolute stasis.

(*b.*) It occasions gangrene by the crushing effect of its products upon the capillaries, or by the mechanical or ulcerous isolation of textural parts.

In the first case, the necrosis affects more immediately the blood held in stasis; in the second, the textures. In the first case the gangrene is, as it were, an immediate issue of the inflammation, the opposite to resolution; in the second it is a remote consequence thereof.

In this way, gangrene may arise in tissues labouring under the sequelæ of inflammation, without being itself an issue of the latter.

3. *Cold gangrene, sphacelus,* is so called, as being unconnected with inflammation.

4. *Moist gangrene* comprises the breaking down of fluid. substances to gangrenous ichor, and of fibrin textures to a variously discoloured, diffluent pulp, marked by its evolution of fetid gases. It is the gangrene developed out of absolute blood stasis;—therefore, again, inflammatory gangrene. It may be compared to the decomposition of animal matter under the co-operating influence of water.

5. *Dry gangrene* is a consequence of deficient blood supply It manifests itself in the perishing of the implicated textures, with shrivelling or withering thereof, to an incipiently tough, but eventually sloughing mass. Often, and particularly in the *gangrene* termed *senile,* which affects the extremities, especially. the inferior, owing to impermeability of their arteries, the gan-

grenous textures blacken; wherefore this species has been designated as *mumifying gangrene*. As such, it is comparable to the decaying of organic matter, that is, to decomposition with absence or insufficiency of moisture, and with the disengaging of pure carbon. Dry gangrene is frequently called *gangrenous slough.*

6. *Black gangrene, gangrenous slough.*

7. *White gangrene, gangrenous slough,* occurs, for the most part, as a consequence of pressure in incarceration; of the denuding of membranous expansions of their subjacent textures, for example, as peritoneal sloughing at the base of intestinal ulcers. Again, it is generated by the necrosis or death of textures replete with fibrino-croupous exsudates, or of such coagulate exsudations themselves. This refers more particularly to the common integuments, the mucous membranes, fibrous and areolar tissue expansions upon wounded and ulcerated surfaces. To this head belongs *hospital gangrene.*

Of these different species of gangrene, several are often concurrently present. Beneath the common integument, often transformed into a swarthy parched rind, in *senile gangrene,* we frequently meet with patches in which the textures are reduced to a humid stinking pulp.

Just as gangrene of the solids, *gangrenous slough,* varies, so in like manner does *gangrenous ichor,* as necrosed blood or exsudate vary, according to the crasis or constitution under which either has become attacked with gangrene. Thus the necrosis of typhous blood differs from that of pus-blood, or of fibrino-croupous blood.

Like normal textures,—diseased textures and new-growths, fibroid, cancerous formations, for example, may become a prey to gangrene. Neither to ulceration nor to gangrene are all textures alike obnoxious. Bony, elastic, fibrous textures resist gangrene more ably than muscle, areolar tissue, or mucous membranes. Lax embryonic textures, as, for instance, certain kinds of cancer, are especially prone to gangrenous destruction.

The constituent elements of gangrenous texture-masses are, more or less well preserved textural débris, larger or smaller black contoured molecules down to a pulverulent granule mass, black and brown pigment granules, fat-drops and crystals, saline crystals.

Contact, reciprocity of action, with the atmosphere, is by no means indispensable to the generation of gangrene. It affects equally with the external parts, organs never in contact with the air, as the liver and the spleen.

A very important phenomenon involving a curative act, is the circumscription of gangrene by an inflammatory process of ulceration,—isolation of the gangrenous part through its own secretion. The ultimate healing is brought about by the same inflammatory process changing to one of pus-production, and of regeneration.

CHARACTERISTIC OF INFLAMMATORY TEXTURES AND DIAGNOSIS OF INFLAMMATION IN THE DEAD SUBJECT.

In the period preceding the real exsudation, an organ is, within the range of the inflammation, reddened, injected; that is, more than ordinarily vascular, swollen, and at the same time relaxed, softer, lacerable. The redness must be that of injection, and is to be carefully discriminated from redness of imbibition. The swelling and relaxation result from infiltration of the texture with exsuded blood-serum.

Generally speaking, the swelling is accompanied by increase of volume; to this, however, spongy textures, and in particular the lung, are exceptions. In protracted, and especially in hypostatic stases, the swelling of the texture frequently occasions closure of the pulmonary cells, and renders them inaccessible to atmospherical air. The volume of the inflamed part falls short of the normal.

As, however, mere hyperæmia occasions similar appearances, although in a minor degree, the question arises: "what are the criteria in the dead body which justify us in pronouncing the inflammatory stasis to be attained?"

The only true criterion in the dead subject is afforded in the changes suffered by the blood in the capillaries of the implicated texture, during the inflammatory stasis—changes cognizable, in part, by the naked eye.

Effusion having taken place, its product, *exsudate,* affords incontestable evidence of inflammation, wherever its character is such as we know by experience to attach solely to the fruits of this process; examples are pus or croupous fibrin. The redness and injection cannot here commonly apply, having, for the most part, given way to the effusion. Even the swollen condition of the texture may in a great measure have subsided during a mortal collapse. The relaxation of the tissues still lasts, taking, however, in solid exsudates, the form rather of fragility, as, for instance, in the hepatized lung.

Where, however, the exsudate is a blastema, well known to exsude both with and without inflammation, the question arises: what circumstances warrant us in assuming such blastema to be the product of inflammation? Such circumstances are—

(*a*.) Rapid and copious production of the blastema;

(*b*.) The concurrence of exsudates, known to be exclusively generated by inflammatory stasis;

(*c*.) Analogy of textural destruction with that due to inflammatory exsudation, more especially if coupled with analogy between the exsudate and a product pertaining exclusively to inflammation, as between tuberculous infiltration of the lung and hepatisation, and again between the hepatizing tubercle and the fibrino-croupous exsudate.

Lastly, it is important to decide whether the appearances, in the neighbourhood of a product, are residue of the inflammation that called it forth, or the rudiments of a new consecutive inflammation caused by the said product. The discrimination, so far as it is feasible at all, may be deduced from what has been already stated.

COROLLARY.

(1.) The inflammatory process is especially fitted for displaying the primitive differences of blastemata, those inherent properties contingent upon internal (endogenous) formative processes.

(2.) It is equally adapted to demonstrate the commonly mixed character of blastemata, and the consequent variety of elementary constituents which enter into the composition of a new-growth.

(3.) It is the last of a series of exsudative processes, beginning with the exsudation of plasma in the act of nutrition, which in degree and kind probably all bear more or less resemblance to it.

(4.) Scarcely a new growth exists, the blastema of which may not be produced by inflammation. On the other hand, inflammation yields products, proper to itself alone. It is to be observed, at the same time, that where a corresponding crasis prevails, the stasis requisite for the formation of the product is in the inverse ratio of the intensity of the dyscrasial process in the general circulation. Such exsudatory acts, together with other processes to be discussed in the next chapter, are wont, owing to the rapidity of the effusion and to the slightness of the accompanying stasis, to be characterised as *deposits*.

(5.) The homœoplastic textures produced by inflammation are areolar tissue, a fibre analogous to that of organic muscle, transition-cartilage, bone, blood, and blood-vessels. The regeneration of nerve-fibrils, after wounds attended with loss of substance, does not take place as a new formation out of the exsudate, but as growth of the nerve from its cut ends into the exsudate, constituting a provisional cicatrix.

(6.) Inflammation possesses now a local, now a general import. In the latter case it is the localization of an anomalous crasis which stands to it in the relation of cause.

(7.) Inflammation may concur with a crasis either accidentally or as its symptom. This has been to a certain extent recognized by the acceptation of an arthritic, a scrofulous, a scorbutic inflammation. Custom has stamped inflammation with fibrinous exsudates and a kindred phlogistic crasis, as genuine inflammation.

(8.) How ought we to estimate that view which designates inflammation as augmented, vital, and formative energy—as increased vascular activity,—as reaction?

Neither in the sense of a neuro-pathological nor of an attraction-theory, can there be any question of increased vascular activity. A vigorous formative power no doubt *is* at work in the inflammatory process; still, in the formative efforts, the qualitative anomaly is predominant. Even adhesive inflammation, in which one might be most of all disposed to look for an augmentation of the normal process of nutrition, produces but few, and these simple, textures.

To define inflammation as a reaction of the organism against a morbific influence, is simply begging the question. Inflammation is a morbid process, unconscious of its scope or object, evoked by a causal impulse, and sustained or repeated so long as this impulse remains in activity. Only in this general sense of cause and effect is the definition of reaction admissible.

DEPOSITS.—METASTASIS (SO CALLED).

Together with those inflammations leading rapidly and insensibly to exsudation, the term deposit (metastasis, capillary phlebitis, lobular process) applies aptly to certain processes which, considering the rapidity with which they become established, are, in many respects, of a very remarkable kind. These processes are founded in a sickening of the fibrin, with a tendency to coagulation. They consist in the blood, through spontaneous impairment or through infection [that is, the reception of various substances], acquiring the tendency to coagulate, and actually coagulating within the circulating system, under a more or less marked separation of fibrin.

This happens either in one of the larger vessels, or, what is far more common, in the capillaries of an organ. In the former case it is, consistently with the frequent reception of deleterious substances into the venous blood, the larger veins, usually in the vicinity of the point of infection. With respect to the capillary system, no point of it is exempt, although the more vascular organs, those in which the blood undergoes important changes, are most obnoxious to the affection; for example, the lungs, the spleen, the kidneys.

In large vessels it is not difficult to interpret the appearances. In the absence of all evidence of local inflammation of the vessel's coats, the caliber is plugged with lengthy, cylindrical, or smaller, clod-like—in vascular trunks membranaceous,—in arteries, especially where the inner surface is rough or gibbous, adherent—coagula. The probability of these coagula resulting from inflammation of the vessels, increases in proportion as the fibrin which constitutes them is pure, and as its yellow colour and general attributes approximate to those of croupous fibrin. The various coagula found within the heart's cavities—the valvular, the globular, vegetations belong to the same class.

The cylindrical and clod-like coagula are often equably reddened by incorporated blood-corpuscles. At other times they contain layers of a lighter colour than the rest, and which therefore have incorporated fewer blood-corpuscles. Some layers are even marked by a total absence of redness, and obviously consist of pure effused fibrin.

To trace the process in the capillaries is not an easy matter. It is conceivable that besides what happens within the vessels, and as a consequence thereof, exsudation of blood serum, and even of a portion of plasma with blood-pigment takes place. By means of this exsudation, the vessels become obscured and uncognizable. Still, the simple fact of the process occurring in large vessels should remove all doubt as to its existence in the capillaries.

In the capillaries of an organ, the process originally manifests itself by a dark red, sometimes reddish-white, spangled or striated circumscribed, impacted substance of a dense, fragile consistence.

These impactions possess the peculiarity of being seated for the most part in the periphery of organs,—the lungs, spleen, kidneys, for instance. They represent roundish tubercula or else wedges with their broad base directed towards the periphery and impinging upon the sheath,—their points towards the interior of the organ. They are always present in considerable number, and commonly associated with exsudative processes. Their size is mostly inconsiderable, commonly ranging from that of a pea to that of a walnut. In organs of a lobulated structure, they are sometimes called *lobular metastases.*

The ulterior changes are various, corresponding with the nature of the coagulation, and therefore of the blood-(fibrin) disease.

They sometimes shrivel and condense, with obliteration of the canals of the vessels, and of the implicated textures, into a fibroid state,—still further shrivelling callus, towards which the neighbouring parts are retracted in a scarlike manner, and which often involves a residue of blood-pigment in the shape of rusty brown, or rusty yellow coloration.

It is probable that, like the thrombus, they are often progressively redissolved, and again taken up into the circulation without detriment to the textures.

Sometimes they break down to a puriform, ichorous fluid, to gangrenous ichor, with diffluence and necrosis of the involved textures, resulting in a pus or ichor depôt, or a gangrenous slough. In greater coagula, this metamorphosis very commonly emanates from the central layer. Capillary impactions, at the periphery of an organ, often assume the aspect of a superficial boil.

The coagula often undergo fatty conversion.

Occasionally, the coagula have the character of tubercle or of cancer, especially of medullary cancer; and it is probably through this process that the often very rapid development of cancer is produced in brutes, by the injection of cancer-blastema.

In conclusion, many an important crase-exhausting metastasis may have its source in the same processes, involving a great extent of capillaries, and issuing in gangrene. Such metastases occur as the sequel to typhus, and to the exanthemata.

With respect to the organs whose capillaries are especially obnoxious to these coagula resulting from infection of the blood, as it is in venous blood that the mischief commonly originates, and as the lungs are the first organs reached by the poison, their capillary tissue is commonly the first to suffer. In its arterial passage, the contaminated blood next attacks the capillaries of the spleen, and of the kidneys. Lastly, in a higher grade of the dyscrasis, all the other textures, mucous membranes, common integuments, and subcutaneous areolar tissue, even bone, become implicated in their turn.

In infection of the portal blood, the liver is the first assailed, otherwise it is only so in common with other organs, and by no means more frequently than the spleen and kidneys.

That, compared with the greater vessels, and especially the arteries, these processes occur pre-eminently in the capillary system, seems due to the slackening of the circulation in the capillaries, which in itself promotes coagulation, and at the same time, leaves the deleterious matter longer in contact with the blood. In certain organs, moreover, their formation appears, as before stated, to be favoured by the revulsion which the blood undergoes in them during the acts of nutrition and secretion.

Sensitive as is arterial blood in respect to infection, as in arteries, for instance, coagula are comparatively rare in arteries of the aortal system, with the exception of those at the point of arterial inflammation. This is probably owing to the great rapidity of the arterial current. In the arterial section of the heart, however, they are readily engendered by diminished action of their organ, far more readily than out of the venous blood in its right chambers.

The relation which these processes bear to inflammation and its terminations, is obvious. Determined by high grades of dyscrasial impairment of the blood, they represent product formation, endogenous exsudation, within the vascular system.

B. ORGANIZED NEW-GROWTHS SPECIALLY CONSIDERED.

Having now treated of organized new-growths in general, of their blastemata, and of the several processes through which these are engendered, we come to the special consideration of new-growths.

What order ought we to observe in this discussion?

1. An arrangement based upon morphological relations is inadmissable:

(*a*.) The elements being in themselves far too uniform, in their secondary arrangement too multiform, and generally too little distinctive of the nature of the new growth.

(*b*.) The elements being mutable, what is one day in the embryonic state, is found further advanced on the morrow; whilst various grades of development are found to co-exist in juxtaposition.

(2.) Similar objections obtain against a division of new-growths according to their main organico-chemical constituents; these being not alone convertible substances, but also liable to enter into various combinations in the same growth.

(3.) With respect to the distinction of new-growths into homœplasiæ and heteroplasiæ, we have already once expressed an opinion. A systematic arrangement upon such a basis, irrespectively of its preternatural rigour, is open to the objection, that it breaks up new-growths into two great series, in the first of which no doubt many points offer in the analogies for further subdivision. In the second, however, we are either driven to a ground of distinction alien to the system, or else compelled, in opposition to the principle itself, still to have recourse to comparison with normal textures.

(4.) How little the benign or malignant nature of new-growths in general, affords a basis for a genuine classification is self evident.

In order to avoid the difficulties alluded to, we shall here endeavour to observe a middle path, and treat of new growths in a series beginning with those which bear the evident tokens of benignancy, and whose ulterior development is for the most part one of progress into fibre; passing next to those differently constituted in these particulars.

1. *Areolar tissue formations.*—The new-growth of areolar tissue is a very widely extended one. Newly generated areolar tissue occurs both pure, and also as a constituent of other new formations, for which it often furnishes a sort of stroma or framework. Generally speaking, it is not alone as to the constitution of, but also as to the arrangement of its fibres in, new-growths, a repetition of the normal. In the said stroma or framework, however, of many cancerous growths, there occur fibrils of extraordinary delicacy.

Its development often follows the laws of the cell theory, that is nucleus, cell-formation, spindle-spaped, caudate cell, fibre with attached nucleus, fusion of several cells to a varicose fibre, breaking up of the fibres into fibrils. Areolar tissue is, however, much more frequently and more extensively shaped out of the immediate dissilience of a solid blastema into areolar tissue fibre, or else mediately, through a preliminary splitting into other coarser fibres.

Newly formed areolar tissue is often found blended in different proportions with elastic fibres, nucleus fibres in various degrees of development, from the oblong, caudate nucleus, the rodlike fibre stem, to the complete fibre.

Apart from its occurrence as mere increment of that previously existing (hypertrophy)—areolar tissue of new formation is met with:

(*a.*) In the shape of threaden, cord-like growths, of flocculent and velvety accumulations, of either free, bridle-like, or agglutinated and firmly seated layers and membranes upon serous tunics, even of entirely new formed, moveable, serous sacs. In many such cases it is, as membrane, invested with an epithelium on its inner free surface.

It determines those frequent adhesions of organs contained within serous sacs, both with each other, and with the parietes of the latter.

(*b.*) It constitutes the entire parietes of perfectly new anomalous serous and synovial sacs, or else it partially enters into their composition for the most part as the external layer, in connexion with fibroid textures. To this class belong the anomalous bursæ mucosæ, the articular capsules of preternatural joints, the capsules investing foreign bodies or extravasate, (the envelopes of apoplectic cysts), every variety of cyst-formation.

(*c.*) It forms the external vascular sheath of many new growths, both benign and malignant, fibroid and cancerous, or their stroma, for example in lipoma.

(*d.*) Of tumours it forms the condyloma, the hypertrophous lupus, the pedunculated wart. Commonly conjoined with fatty texture, it composes those appendices of the skin denominated mollusca, a species of so-called fibrous tumours and of fibrous polypi.

(*e.*) Finally it presents in various grades of development an essential constituent of scar-texture.

The growths composed of it contain a large proportion of gluten.

The blastema for the new-growth of areolar tissue is sometimes fluid, and the devolopment takes place according to the cell-theory laws, but more often, especially when copious, it is solid and fibrinous. It exsudes during the progress of protracted hyperæmiæ, and in the last mentioned form, more especially, as a consequence of inflammatory stasis. Both modes of development concur with suppuration. Moreover, extravasated fibrin, as also endogenous fibrin-coagula within the vessels furnish, under certain conditions of the fibrin, the blastema for the new formation of areolar tissue.

The chemical changes which take place during this process of development are very remarkable. They consist in numerous modifications of quantitative type, as also in qualitative differences of reaction observable in the gluten-extracts.

The time requisite for the new growth of areolar tissue varies from one to several weeks. The process of dissilience into areolar tissue fibres and fibrils, more especially in the case of copious solid blastemata, is often a very slow one.

2. *Fibroid texture.*—In an extended sense, the collective term *fibroid texture* may be made to comprise all fibrous tissues, the development of which has been already delineated, and the

occurrence of which as a more or less essential component of various new-growths, it becomes our business to discuss.

Nowhere is the insufficiency of a mere anatomical principle more felt than here—a principle which would needs occasion us to class side by side, the most heterogeneous new-growths, for example, fibro-carcinoma and the perfectly benign fibroid tumour.

In a more restricted sense we comprehend under fibroid textures those which consist either of the elements about to be described or of a blastema manifestly furnishing their groundwork, which yield gluten, are benign, and in external appearance, resemble the fibrous texture. In this sense the fibroid texture enters into the composition of various heterologous growths, constituting in them the benign ingredient which cornifies or ossifies by a spontaneous metamorphosis. Or it is woven in with normal textures, or, lastly, it represents sharply defined, often very voluminous masses, in a word, tumours. The fibroid (fibrous) tumour, besides the aforesaid attributes, is distinguished as consisting entirely or almost entirely of the elements of the fibroid texture. It is firm and elastic, or else tough, presenting a mere local evil, independent of dyscrasial taint and originating in local deposition, a fact denoted more especially by its selection of a particular organ, even where it occurs in great numbers.

The groundwork of fibroid new-growths are firm, probably always fibrinous blastemata. Exsudation or extravasation-fibrin, or fibrinous coagula within blood-vessels often constitute these.

Irrespectively of perfected areolar-tissue-fibre, as the main, if not the sole constituent of growths called, from their dense texture and their resistance, *fibro-areolar textures, or fibrous tumours,*—the following elements are in particular deserving of notice.

(*a.*) Flat, smooth fibres resembling the organic muscle-fibre, here and there breaking up into fibrils, and thus engaged in the transition to areolar tissue. The nuclei present deport themselves as upon muscle-fibre, and the textures proper to it.

(*b.*) Flat, broad, band-like or roundish, shapeless, solid fibres, with rough, denticulated or felt-like outline, which are held together by a solid blastema, and here and there break up into areolar tissue fibrils. Nuclei often seem to stand in no developmental relation to them, and are frequently altogether absent.

(*c.*) The *fibroid blastema*, an embryonic, stratiform, fibro-laminated, solid, transparent, or opaque (brawn-like), formation, interspersed, or not interspersed, with elementary granules, nuclei, and cells.

The delicate fibre network of solidified fibrin is occasionally preserved in it.

The above-named fibre-elements originate directly out of their blastema through dissilience. The formations consisting of them occur both as superficial expansions, and interwoven in normal textures, as knotted, spherical, or irregularly ramified masses (callosities); and, lastly, as independent tumours.

These various formations demand a special enquiry. Besides the true fibrous tumours, of which we shall have to treat specially, we have here to mention:

Inflammatory products, fibroid exsudates, representing within parenchymata, irregularly knotted, ramified masses, or, upon serous membranes, superficial expansions. The latter constitute pseudo-membranous calli of various dimensions, but frequently engrossing the entire superficies of a serous sac. They are of various thickness, which is considerable upon the parietal layer of serous sacs, of density and resistance equal to those of fibro-cartilage; in colour white, or, as a consequence of hemorrhagic exsudation, varied with black, slate-gray, rust-brown, or a yeast-yellow. They often determine complete conglutination of the parietal and visceral layers of serous sacs. Or, again, they are smooth and even, or fenestrated, granular, stellate plates; or finally, they consist of granulations, scarcely surpassing in size, poppy, millet, and hemp seeds, for example, upon the cerebral arachnoid membrane, upon the hepatic and splenic peritonæum, &c.

The fibroid thickening of serous membranes in the shape of smooth or granulated plates, of granulations, to which last are to be reckoned the Pacchionian glands.

Most free bodies found within serous and synovial sacs.

Cicatrix-substance generally, as also the so-termed keloid of Alibert—arrested (ligamentous) callus.

The internal layers of numerous cysts and of receptacles and excretory ducts degenerated into dropsical capsules (dropsy of the gall-bladder; of the Fallopian tube).

Callosed extravasate-fibrin, in the shape of central or peripheral (encysting) membrane separated out of extravasates.

Callosed coagulations of fibrin within the vascular system, the different so-called vegetations in the heart's cavities, the cylindrical coagula within veins and arteries.

The superimposed layers upon the internal surface of arteries; the soft ground-work of so-called phlebolites; &c.

These fibroid formations not unfrequently inclose within a nidus, a curd-like or puriform fibrin—the product of inflammation.

The secondary arrangement of the elements above treated of, is reducible to the following types:

1. *Parallel fibrillation,* superficial expansion predominating.

2. *Fibre-felt,* a multicrucial fibrillation, a section of which, in whatever direction made, always displays fibre-shoots and bundles, intersecting each other at various angles.

3. *Areolar disposition,* of very rare occurrence, at least in pure fibroid formations springing from a solid blastema.

The two following structures, determined by a primitive disposition of the coagulating process, are also rare.

4. A network of fibroid bridles (fibre bundles), of from $\frac{1}{80}$th to $\frac{1}{13}$th of a millemeter broad, crossing each other at various angles, and having their gaps filled up with embryonic elements—for the most part nucleus-formations in an amorphous blastema.

5. A web of similar fibroid cords emanating from a central mass, and anastomosing with other webs derived from other centres.

These structures seem to occur more especially in fibroid formations springing from extravasate-fibrin, as occasionally met with in stratified deposits upon the inner surface of arteries.

6. Finally, a kindred form is brought about by resorption, as a gap- or fenestrate-formation, the gaps being round or oval. It occurs in fibroid tumours, in the fibroid thrombus, in the vegetations within the heart's cavities, but especially in the accumulated layers upon the internal arterial membrane. It is analogous with the fenestrated structure of the striated coat of blood-vessels.

All these formations have, even for the naked eye, the aspect of a porous, cancellated structure; the gaps are, however, widely different from the alveoli of areolar textures.

(*d.*) A further element of fibroid formations is a cylindrical, in its parietes structureless, striated fibre, with a simple, but sometimes a double contour, inclosing granules, nuclei and cells. We have encountered this sort of fibre in consolidated hemorrhagic effusion; in old vegetations about the heart's valves; in fibred cartilaginous investments of the joints; in the villous new-growths upon synovial capsules.

(*e.*) Another element, again, of fibroid growths is the nucleus and the nucleus-fibre in the intermediate stages, as caudate nucleus, and varicose nucleus-fibre. These elements are to a certain extent found in conjunction with those hitherto discussed, with the concurrence, however, of an amorphous bond-substance—a membranous basement. They extensively furnish forth fibroid growths, but are not very common.

(*f.*) To conclude, not a few fibroid new-growths consist in a fibre-felt, developed within a basement either solid or adapted for membranous expansion. It resembles that in the intercellular substance of reticular cartilage. This texture is often met with in the fibroid deposits within arteries.

A metamorphosis common to fibroid textures, is a so-called ossification, and a cornification. The former is observed more especially in fibroid tumours, in fibroid exsudates, upon serous membranes, in fibrinous coagula within the caliber of blood-vessels, in the deposits upon the inner membrane of arteries, and the like. The growth shrivels with obliteration of its vessels, loses its elasticity, becomes dry, of a dingy yellow colour, and gradually bereft of its fibrous texture. Meanwhile a black, minute molecule, consisting of fat with the salts of lime, becomes imbedded in its substance.

Cornification is observed especially in the vegetations about the heart's valves, and in the layers accumulated within arteries. The growth becomes dry, denser, of horn-like toughness, and of dull transparency.

Vascularized fibroid growths occasionally take on inflammation, for the most part from the surrounding tissues, suppurate, and perish layer by layer. Nor is it rare for them to become loosened and cast away through the suppuration of adjoining textures.

Gluten yielding fibroid tumour, also denominated fibrous tumour, formerly designated as sarcoma, steatoma, or even as scirrhus.

They are distinguished from other fibroid tumours by their independence and circumscription, as being sheathed in a layer of vascular and areolar tissue, and thus, as it were, impacted in the texture of organs, from whence they may be fairly peeled out. They represent more or less perfectly spherical, for the most part tough, fibro-cartilage-like, resisting, distinctly fibrous, according to their degree of vascularity whitish or reddish-white, new-growths. In size they vary from that of a tumour just cognizable, up to, and beyond that of a man's head. They often coexist numerously in the same organ.

They probably occur in every organ, although they are, without doubt, frequently confounded, more especially in glands, with fibrous sarcoma, and with fibro-carcinoma. They are frequent in the submucous areolar tissue layers, more particularly in the intestine, stomach, and œsophagus, now and then in the larynx, again, in the subcutaneous areolar tissue, and very commonly in the uterus and its appendages, where their development is in every way very strongly marked. Their texture is usually made up of areolar tissue fibre, or of the elements described at *a*, *b*, and *c*. The uterus fibroids, in particular, very often repeat the fibre of organic muscle tissue.

With respect to the arrangement of the constituting fibres, fibre-layers and fasciculi, much variety is manifested, and this, again, is pre-eminently marked in the uterus fibroid. Thus:

(*a*.) The fibroid tumour, with *concentrical lamination of the fibre-layers*, is commonly quite spherical, very dense and tough, poor in blood-vessels, white, never attaining the magnitude of the species that follow.

(*b*.) Fibroid, with manifold *decussation of the fibres*. The fibration frequently springs from distinct centres marked by their density and whiteness. These tumours grow to a considerable magnitude, and have an irregular flattened tuberosity of surface.

The following is a variety of this fibroid:

The tumour consists of an *aggregation of dense fibroid tubercula or centres, about the size of peas or beans, united by means of a lax vascular texture.* These tumours have an uneven undulating surface, and attain to a very considerable bulk. The loose interstitial texture sometimes becomes the seat of a serous infiltration, which, under dragging and eventual laceration of the said texture, may become exalted into dropsy within the tumour. The fibroid contains within its interior a cavity replete with serous fluid, fluctuates, and may thus present the appearances of a cyst—in the uterus, more particularly that of a hydrometra.

All these fibroids ossify.

(*c.*) There is one other variety of fibre-tumour, which, so far as its elements are concerned, ranks with the foregoing growths. In other respects, however, it differs from them, and offers a transition form to fibro-sarcoma.

The characteristic of this last form of fibrous tumours is that, varying in circumference, they are so rooted in the implicated organs as not to be removable from, without injury to, the latter; that they become lobulated in their growth, and yield little gluten; whilst, on the other hand, they contain albumen, and in their fibrillation are, at least in part, developed out of cells. Not rarely we find in them excavations lined with a smooth membrane, and filled with a sero-albuminous fluid, *alveoli, cysts.* They are further marked by considerable vascularity.

Like other kindred forms referable to the class of fibro-sarcoma, they are often rooted in the sub-mucous, areolar, and muscular textures, in areolar tissue, in the periosteum subjacent to mucous membranes, and in the inner layers of the substance of the womb. Here, under the designation of *fibrous, sarcomatous flesh-polypi,* they grow into the muco-membranous cavities, with predominant longitudinal direction of their fibres, pushing forward the mucous membranes themselves in their advance, and representing cylindrical, spindle-like, pear-shaped, bulbous tumours, lobulated at their free extremity, and traversed, more especially in the uterus, by capacious blood-vessels (veins).

As so called polypi, they are to be carefully distinguished from mucous, cellular, or vesicular polypi.

They do not ossify.

3. *Elastic tissue and texture of the annulo-fibrous membrane of arteries.*—The elastic and nucleus-fibres enter, more or less, and sometimes in very considerable quantity, into the composition of the most varied new-growths, although in no instance are the latter entirely composed of them.

We have, however, occasionally observed accumulations of elastic fibres, in the arrangement and form proper to the vocal chords, beneath the mucous membrane of the trachea, and close to the larynx.

With reference to the texture of the annulo-fibrous membrane, we have, in one instance, seen the muscular fibres of a hypertrophied, solidified, rigid, muscular tunic of the urinary bladder converted into dingy yellow, elastic bands, which presented a texture exactly like that of the annulo-fibrous membrane of arteries. A transformation of one texture-species, pertaining to a common genus, into another.

4. *Cartilaginous growths.*—Wounds of cartilage are not reunited by means of cartilaginous substance, nor is this substance regenerated when destroyed. Nevertheless, new-growths of cartilage-texture are both frequent and voluminous. The structure of these growths or tumours was first ascertained, with the aid of the microscope, by Johannes Müller, who applied to them the term *enchondroma*. These excepted, not a single new-growth, whether designated as cartilage-like, fibro-cartilage-like, or as cartilaginescence, chondroid, fibro-chondroid, has more than a seeming analogy with true cartilage texture.

Enchondroma repeats all the special physiological textures of cartilage. It occurs both as hyaline, genuine, as fibro-cartilage, and as reticular cartilage, imitating the articular investments, the laryngeal cartilages, and the septum narium on the one side, and the synchondroses of the vertebræ, the cartilages of the external ear, the epiglottis, &c., on the other.

Ordinarily, and especially in the enchondroma of soft parts, all these forms are often found in juxtaposition. The pure hyaline cartilage is, however, the least common, the intercellular substance displaying, for the most part, a fibrillation similar to that in the cartilages of the ribs.

The *enchondroma* forms spherical, or nearly spherical tumours, with an even, smooth, or else, which is more usual, a mammillated surface. Internally it either presents a continuous hyaline mass, or else, corresponding with its mammillated exterior, a lobulated structure, an aggregate of denser, hyaline knobs or spheres, either held together by a black contoured, rough, inelastic fibre texture, resembling the intercellular fibrillation, or else imbedded in a loose texture imitating the fibre-layer of reticular cartilage.

Enchondroma chiefly occurs in bones, especially in the phalanges of the fingers and toes, in the sternum, in the ribs, more rarely in other bones, such as the long cylindrical bones, the ilium, the skull-bones. It is also met with in the mammary gland, in the parotis, in the testicle. We have ourselves seen it in the subcutaneous areolar tissue, and on several occasions in the lungs.

In magnitude, enchondroma varies from that of a tumour only just cognizable to that of a child's head, and beyond it. In bone, enchondroma exhibits two varieties, namely, enchondroma *with*, and enchondroma *without* bony sheath. This osseous capsule is bone, whose texture has become distended and inflated by the enchondroma in the progress of its upward development. In this process it has, for the most part, increased in substance, so that the capsule far exceeds, in this respect, the original bone. Where the sheath ruptures at an early period, the enchondroma is devoid of bony investment. The capsular case of the enchondroma is unessential, and is common to many other heterologous growths developed out of the depth of bone, and more especially out of a medullary cavity. Many of the so-termed cases of spina ventosa of older observers, were probably of the nature of enchondroma.

Enchondroma is benign, provided it does not enter into any specific, infectious metamorphosis, and only undergoes ichorous destruction from irritation. A peculiar predisposition to its formation does, however, exist, as shown by its occurring numerously in one individual, (phalanges, ribs, &c.) It affects young persons more especially, although we have known examples of enchondromata first becoming developed at an advanced period of life. Here, however, they are usually concurrent with exostoses and bulky osteophyte forms. Echondroma generally imitates the permanent cartilages; with exceptions, however,—for it ossifies.

Not only have we seen in all enchondromata incipient ossification, but our museum contains specimens, for the most part, if not thoroughly ossified.

Ossified enchondroma is sometimes a white, extraordinarily dense, ivory-like, sometimes a yellowish-white, likewise very dense, although uncommonly brittle, bony substance, deviating in various degrees and various ways from the texture of normal bone. This difference of habit corresponds to a different process of ossification, and to a different elementary texture.

In the first place, we miss the laminated structure of true bone. The medullary canaliculi are present, the bone corpuscles large, spherical, irregularly grouped, wanting in radiations.

In the next place, and this refers to the second form of bony substance adverted to, the process of ossification recedes still further from the normal. It resembles rather a process of involution, a wearing out of the cartilage, and, like the texture itself, it has its analogies in ossification of the larynx, and above all, of the cartilages of the ribs. The intercellular substance of the hyaline enchondroma becomes dull, granulated, sallow, lardaceous, and fibred. The cells are centrally transformed into spherical bone-corpuscles without radii, or else the entire large cell-cavities are simultaneously, if not previously, filled with bone-earth. They are interspersed without order, the last mentioned forming very comprehensive spherical or oval masses, which, with transmitted light, appear black, and and have a diameter of $\frac{1}{15}$th of a millemeter. The lamellated structure is wanting. Medullary canals are wanting, or rudiments only of their structure are seen in the scattered grouping of the cartilage cells.

Enchondroma commonly occurs in a simple form. We have, however, encountered it in the shape of little millet or hempseed-sized tubercula, interspersed through medullary carcinoma of the testicle, an occurrence allied to the frequent entering of true bone into the composition of cancers.

5. *Bone-formation.*—Bone-formation comprises various new-growths, which, in their developed stage, are readily divided into two classes, according to the analogy which their texture bears to that of normal bone. Still the line of demarcation is not sharply drawn, owing to the multiplicity of transition forms from the one to the other. The one category comprises new-growths *identical, or nearly identical with,* the other, *a series of new-growths less or more widely discrepant from the texture of normal bone.* On a closer scrutiny, however, this series again separates into the *osteoid,* and into the *bony concretion,* which latter manifests itself, more especially in fluid blastemata, as *cretefaction.*

It is, indeed, worthy of preliminary remark, that not alone solid blastemata and perfected textures, but also fluid blastemata, afford the basis of, and are liable to, so-called ossification.

The process which involves the conversion of the substances here alluded to into bone, is commonly termed *ossification*, and thus brought into kindred relation with the bony conversion of cartilage.

We have here to observe:

1. In the first place, amongst the pathological growths with which we are here more especially concerned, are ossifications for the most part not constructed upon a preformed cartilaginous base, whilst many of them deviate from the course and the results of the ossifying process of bone-cartilage. It will be seen that in these last referred to, there is an absence of the vascularization proper to the cartilage in its transition to bone, an absence of that lamellated structure-development, with that grouping of the cartilage-cells, and that resulting arrangement of the bone cells, which both exhibit in common. In isolated cases, as, for instance, in the ossifying of enchondroma, it is not in the intercellular substance, but in the cartilage cell, that the ossification first commences. That ossified enchondroma differs in essential points from the texture of true bone, we have already seen.

Where no preformed cartilage, but rather a rude, firm, sod-like, and fibrous, or a fluid blastema, or, again, an anomalous, mostly fibroid, texture, constitutes the groundwork of the ossification, the result is a concretion more or less uniformly penetrated by bone earths, and presenting scarcely any analogy with the texture of bone.

2. The characters of ossification differ according to certain differences in the implicated textures; or, where the textures are identical, according to certain peculiarities in the process itself.

The ossification of a cartilaginous base has frequently, although not always, an import coequivalent with that of ossified bone-cartilage—in other words, the import of a progressive metamorphosis into a complex vascularized texture. Genuine bone-texture, on the other hand, can, in the present state of our knowledge, be traced to a cartilaginous base alone, the pre-existence of which, if not obvious, must be taken for granted.

The ossification of other textural bases, on the contrary, has, if we take into account other collateral changes which these bases, and more especially the fibroid textures, undergo, the import of a retrogressive metamorphosis, of a decay, of a destruction of the base. With the display of lime-earths in the shape of black molecules, the textures lose their colour, their succulence, and their elasticity, waste, shrivel, toughen, dry up, and become more or less lardaceous. Not alone do no new blood-vessels make their appearance, but old ones, if there be any, become obliterated. Fluid blastemata in the course of cretefaction become turbid, chalky, gritty to the feel. Under the development of fat, they form into a pap, and eventually thicken down to a mortar-like concrement. Even in cartilaginous bases, ossification is often so modified as to represent rather a retrogressive than a progressive metamorphosis (compare enchondroma).

3. A most important and comprehensive question relative to ossification generally, and therefore to bone-cartilage inclusive, is: whence are the lime-earths which incrust and penetrate the various soft textural bases derived?

A narrow scrutiny of the ossifying process, especially in fibroid textures, and of the cretefaction in soft and fluid blastemata, will speedily convince us that the appearance of lime-earths is not essentially due to their deposition out of either a pre-existent or a new formed and special system of blood-vessels within the ossifying growth. For, when we see growths ossify, which are almost, if not entirely devoid of blood-vessels, and which are, at the same time, remote from the vascular system of other formations (for instance, free bodies within serous sacs); when we see the process of ossification often attended, not with any new growth of blood-vessels, but with the obliteration of existing ones; when, again, we reflect upon the concomitant changes wrought in textures during their osseous conversion, their wasting and discoloration, the interlarding of their shrivelling substance with free fat, we are fain to look upon the entire process as the result of the total transformation of the chemical constituents; as, in fine, an elimination of pre-existent lime-earths out of their primitive connexions.

Even in the normal ossifying of bone cartilage, the process is the same at the commencement, the lime-earths appearing long before the development of any vascular system. This, then, offers at least one connecting link for all processes of ossification.

In the revolutions effected by the ossifying process, a most important part is without doubt assignable to the accession of fat. It is common to all processes of ossification, and probably results both from a release of pre-existing fat from its primitive combinations, and of a simultaneous conversion of protein substances into fat.

From these preliminary remarks, we may at once proceed to a muster of the new-growths belonging to this category, premising, however, that much relating to them will have to be discussed more at large in later chapters of the present work.

1. Uniform or almost uniform with the normal bone, are:

(*a*.) Bone developed in permanent cartilages, and especially in those of the larynx, sometimes and in part also the ossifications of costal cartilages. In them, however, we usually miss the lamellated structure of normal bone.

(*b*.) Bone-structures which form as callus for the reunion of fractured, and for the regeneration of lost bone, hyperostosis, whether external or internal (sclerosis), exostoses, and all osteophytes, including such as in the shape of thorny, stellate, or scaly bone-growths and fabrics, enter into, and sometimes greatly surpass in volume, certain concurrent new-growths, especial such as occur in bone.

Notwithstanding the all but identical relations of the texture of these formations with that of normal bone, they present not a few important discrepancies, cognizable both by a general comparison with normal bones, and by a special comparison with those directly implicated. Thus, as examples, we may adduce the inferior vascularization, inferior number of medullary canals, less marked lamellated structure, anomalous amount and irregular disposition of the bone-corpuscles, in the new bone-growths.

As regards the process of ossification in the several blastemata, that produced by inflammation is the best adapted for investigation, as being at once the most frequent, and the most voluminous. The flaky or fibrous basis of the exsudate furnishes the fundamental (intercellular) substance of the cartilage.

Within this cells become developed, which, following the process of physiological bone-formation, change into bone-corpuscles.

(*c.*) The slowly developed bone-nuclei in callus, arrested at the stage of a ligamentous formation in bone fractures, trephine-gaps, &c.

(*d.*) Osseous growths developed beyond contact with bone on the dura mater, as also upon the cerebral arachnoid, and upon the free visceral plate of the spinal arachnoid membrane; the so-called ossifications upon the intermuscular ligaments in the vicinity of hyperostosed articulations, and of the membrana obturatoria of the pelvic foramen ovale. The ossifications occurring in tendons are said by Henle to be of true bone-texture, as are also, in fine, the bony concrements found impacted within healthy muscular textures.

2. *Osteoid.*—Several of the growths adverted to as deviating in certain points from standard bone-texture, might be transferred to this section. To it, however, belong more especially ossifications of costal cartilages, and most of all ossifying enchondromata, both in bones, and in soft parts. In bone there occur independent texture-supplanting tumours, which consist of an ivory-like, dense, white bone substance, and which are seen both in this, and in their general character to be ossified enchondromata.

Müller's osteoid is a bone-formation which enters redundantly into the parenchyma of cancer. Its constitution is identical with that of true bone, and it will be discussed under the head of cancers.

3. *Concretions.*—Under certain, as yet unknown, conditions, the lime-salts in a soft basis are, by dint of a revulsive metamorphosis, set free so as to incrust and penetrate the said basis,—in a word, effect its ossification.

Such bases are the fibroid textures and blastemata occurring in the form of independant tumours, of membranous expansions, or of irregular masses lodged and entangled within various parenchymata. They may be the product of inflammation (exsudation), or merely of an anomalous act of nutrition. The blastema may moreover be extravasate-fibrin, or spontaneous fibrin-coagulate within the vascular apparatus. Such bases are fibrous tumours, fibroid hardened exsudates upon normal and anomalous serous membranes, within parenchymata, in the cutis as scar-texture, on the heart's valves, in muscles, in the heart's walls. Such, again, are the central and the peripherous fibrinous deposits in extravasates, after entering upon a fibroid transformation, hypertrophous thickenings of serous membranes and of the tunicæ albugineæ. Such are, in fine, the Pacchionic bodies, the different fibrin coagula in the heart's cavities (so-called vegetations), the stratiform deposits within arteries, and the soft matrix of the phlebolite in veins. To sum up, therefore—all the so-termed ossifications of serous membranes, of the thyroid gland, of the heart's valves, of fleshy muscle, of arteries and of veins.

Even the fibroid fabric which enters into the composition of malignant growths, for example of cancers in soft parenchymata, now and then ossifies into a bony skeleton, or shell-like framework. This is, however, not to be confounded with the thorn-like, stellate and scaly stroma of true bone-texture, accompanying numerous heterologous growths as developed in and upon the bones.

The ossification offers little or no analogy with normal bone, and its development. The bone-earth enters in a molecular form, accumulating, for the most part irregularly, in the soft basis, until the latter is converted into compact bone. The bone-earth is capable of being withdrawn by acids, with restoration of the soft basis. It has sometimes acquired the aspect of a stratiform deposit. Many a wide-spreading ossification,—of the arteries for example,—is concurrent with excessive fat-production in its vicinity.

4. *Cretefaction.* Finally, fluid blastemata are also liable to ossification. The process is perfectly identical with that which takes place in the fibroid blastemata just enumerated. It is in like manner conditional upon a metamorphosis of the fluid blastema, by virtue of which, the incorporated lime-earths being set free, predominate. Morphologically speaking, the blastema displays the development of free or of celled molecules (granule-cells.) It is always accompanied by fat in a molecular form, and by cholesterine crystals. The ossification manifests itself as a lardaceous chalky pulp, as a cement-like and friable, ultimately, as a compact calculus-like growth.

The blastemata entering into this process are either originally fluid, or else originally solid, and subsequently liquefied (croupous fibrin).

These blastemata are either exsudates external to the vascular system, or deposits internal to the latter, for instance, accumulated layers within the arteries, the basis of vein stones in the veins, or coagula smaller or greater in extent (vegetations).

To this series belong the cretefactions of fibrinous, albuminous exsudates, of pus, of tubercle, of atheroma in arteries, of vegetations on the heart's valves, of coagula in the veins. Just like ossification, cretefaction presents various grades from the aforesaid progressive pulp-like thickening, to the cement-like, and at length the compact, calculous concretion.

A very peculiar kind of ossification is represented in the *cell-incrustations*, which have their physiological analogues in the pineal concretions. They appear in a variety of shapes in the vascular plexuses, especially in the turbid, chalky, speedily condensing moisture of the cysts of the choroid plexus, as also in sarcomata and cancers, more especially within the brain. The cells, both as primary and as parent-cells, together with their contents, fill with lime salts, now in a molecular shape, now in that of concentrical layers.

6. *Growth of blood-vessels.*—Setting aside all dilatations of the smaller vessels and capillaries, constituting the so-called aneurysma anastomoticum and telangiectasis, we shall here treat of all that concerns the new-growth of blood-vessels.

Upon a somewhat slender foundation of facts with which we have to commence, we must endeavour to build up a superstructure of well-considered hypothesis.

1. The common occurrence of this new-growth in inflammatory products is incontestable, more especially in the adventitious membranes, affecting serous tunics. It is at the same time matter of certainty that such new vessels by no means originate through any prolongation of pre-existent vessels in the contiguous textures, but that the new process of development is altogether an independent one, and that only at a later epoch do the new-formed vessels enter into anastomosis with the older ones.

The process of the new-growth of blood-vessels in inflammatory products of this kind is as follows:

In the first place, blood is seen to occupy little vacant spaces within the blastema or exsudate thrown out in consequence of the inflammatory stasis. In other words, certain, for the most part irregularly spherical, ramified, to the naked eye point-like, areæ unlined with any proper membrane, fill with blood; areæ obviously determined by the upspringing of blood out of the blastema. Both their remoteness from old vessels, and their ulterior development controvert the idea of the contained blood consisting of little extravasates. This blood represents an aggregate of blood-corpuscles which the concurring testimony of Vogel and myself has shown to be of various magnitude, for the most part imperfectly round; not precisely disc-like, nor possessed of the intense redness of old blood-corpuscles. They are moreover soft, and adhere both to each other and to the parietes of the containing areæ. From these areæ there are gradually developed in all directions, sometimes, however, predominantly in a single one, blood-streamlets contained within chinks or canals in the blastema, and having no perceptible confining membrane. In minuteness of calibre, they excel the finest capillaries. Their next step is to become invested with a structureless confining membrane, the internal blood-vessel membrane, to which the outer layers associate themselves by and by. Finally they shoot out from various centres to anastomose with one another, and eventually with the old textural vessels.

In this process two material points remain unaccounted for, namely, first, the primitive development of the blood-corpuscles in those central areæ. The question is—their spontaneous and independent origin out of the common blastema being manifest, what portion of the latter is devoted to this purpose, and what chemical changes does the act involve?

Secondly: How do the vessels originate? In reference to the last question it may be asserted:

(*a.*) If we may judge from past observations, the vessels in these blastemata do not spring from primary blood-holding cells. The absence at first of any sharply defined contour in the said areæ disproves their being primitive cells, or the blood-streamlets emanating from them, prolongations of cells. This leads us to the more probable assumption that:

(*b.*) The blood forms out of the blastema generally, and where the latter has wrought itself into cells, not within these, but in intercellular spaces between them. Where, however, blood and blood-vessel formation frequently precede cell development, and more especially where it takes place in blastemata in which cell-development is either wanting altogether, or plays only a very subordinate part, (for example, in fibroid blastemata), it can only be alleged that the formation takes place in chinks and chamberlets worked out of the blastema by the blood itself. The structureless membrane which subsequently confines the blood streamlets, is in all likelihood a secondary endogenous formation out of the blood, the remaining layers being built over it, so to speak, externally, out of the blastema.

As regards the relation of new blood-vessel development to the character of the blastema, the following points seem to present themselves for further inqniry.

(*a.*) In different products of inflammation, there is virtually a very different degree of proneness to the new-growth of blood-vessels. In some it is excessive, in certain others, in the progress of development into precisely the same texture (the areolar), it is very faint indeed. Upon the parietal layer of the cerebral arachnoid, adventitious membranes occur, which along with an inconsiderable amount of nuclei, of nucleated cells, and of areolar-tissue fibrils, consist mainly of blood-vessels.

(*b.*) The new-growth of blood-vessels is directly proportionate to the general capacity of the blastema for textural change. Blastemata which linger long in their primitive crude condition, and also which serve for the ground-work of indistinct fibroid textures, exhibit little, if any, new-growth of blood-vessels.

The new formed vessels give token of a delicacy of structure, and a vulnerability in full accordance with their recent origin. To this are probably due the hemorrhagic products upon their becoming, when only just formed, the seat of inflammatory stasis. The fact is, moreover, to be inferred from the new formed vessels originally consisting of the primitive vessel membrane alone; and not until a later period attaining the perfected organization of old blood-vessels. In new formed vessels of considerable calibre, we have frequently missed the layer of transverse-oval nuclei.

The vascular apparatus of new-growth is marked by, for the most part, long, stretched vessels, by rare dichotomous ramification, with hardly perceptible decrease of calibre, or by a wide-meshed disposition.

A nervous system has not as yet been demonstrated in them.

The new blood-vessels both in the spurious membranes adverted to, and also in other new-growths, are, as experience has again and again shown, susceptible of inflammation, and this in very early stages of their development. Here, for reasons both self-evident and already referred to, they are wont to yield essentially hemorrhagic products.

The manner in which their anastomosis with old vessels, or with particular arteries or veins is effected, has not been thoroughly made out by experiment. As regards the former case, it is probable that the anastomosis is brought about by an act of resorption in the wall of the old vessel, at the point where the *new* vessel rests.

With respect to the second point, Van der Kolk has distinctly shown that in the case of adhesions of the lung to the costal parietes, a connexion becomes established, on the one side with the system of the pulmonary artery, on the other side with the aortic circulation. And although this fact may not warrant all Van der Kolk's deductions, it is nevertheless highly important, as offering a connecting link with other observations of his own upon anastomoses, to which we shall hereafter have to revert.

New-formed vessels doubtless undergo obliteration in spurious membranes, just in the same gradual manner as in the cicatrix, and probably for the most part in the progress and sequel of the retro-gression,—the wasting—of the new textures themselves.

2. As in inflammatory products, so also in other blastemata, does a new-growth of blood-vessels occur. An examination of these proves incontestably that there is a second mode in which a new growth of blood-vessels may take place. We have satisfied ourselves of the *development of new vessels out of parent-cells in cancerous structures,* composed, amongst other elements, of spherical or acinus-shaped parent-cells. Numerous cyst-like cells, with structureless parietes, contained in place of the brood cells of carcinoma, soft, adherent blood-corpuscles. Many of them bulged out in all directions into cœcal sacs, freighted with the same contents, and entering into anastomoses with others. Blastemata, therefore, entering into cell-development, are in reality capable of producing a new development of blood-vessels out of cells.

Certain new-growths are remarkable for being rich, others for being, in various degrees, poor in blood-vessels. The former are mostly loose in texture, and consequently capable of considerable intumescence. Their hyperæmiæ are for obvious reasons, readily exaggerated into hemorrhage, and their inflammation is especially liable to determine hemorrhagic products. Amongst the malignant new-growths, medullary cancer is notorious in this respect. Its highly developed vascular apparatus is doubtless the source of its excessive nutrition, of its rapid and often monstrous growth. It is pronounced by Van der Kolk to be of arterial nature, in other words, to anastomose with arteries only.

Very vascular new-growths form the transition link to nearly pure blood-vessel formation. For blastemata are not wanting which present almost exclusively the ground work of new blood-vessel formation—in other words, of a new-growth, consisting almost exclusively of new blood-vessels.

To this class belong the following new-growths, concerning the development and import of which much obscurity still prevails.

(*a.*) *Cavernous textures, cavernous blood-tumours.*—These growths are of a cancellated structure, somewhat resembling that of the corpora cavernosa. They consist of areolar tissue fibres, constituting a multilocular stroma, the interspaces of which are invested with a structureless membrane, and contain blood. Numerous caudate cells liberated during the investigation appear to be the débris of an epithelium. The intercellular spaces communicate amongst each other; for, by pressure, the tumour may be completely emptied through a cut surface. They are surrounded by a tolerably dense capsule of areolar tissue, along with which they may be peeled out of the textures. They always communicate with a considerable vein, through which they will take up an injecting mass. No arterial ramification is demonstrable within their texture. They are very tumefiable, forming upon the surface of the body and at the periphery of organs, protuberant, compressible, but resilient dark-blue tumours, which supplant the original textures.

They are commonly regarded as telangiektases, and without doubt they pass, with many practitioners, for examples of fungus hæmatodes. By the French (Andral), they are termed "spurious spleens" (Aftermilzen), "placenta-like textures." In our opinion, they are by no means dilatations of blood-vessels, but to all intents and purposes new-growths, and, as far as our experience goes, altogether benign. We have never seen them concurrent with a malignant growth.[1]

Their mode of development is not clearly made out. Numerous observations, however, render it probable that they originate as blood-vessel growths in their most extended sense, as blood-bearing depôts and canals, formed by absorption out of a solid blastema. Whether the contained blood be a primitive endogenous new-growth, or become introduced into it subsequently, we know not. Thus considered, they bear a close analogy with the fenestrate and canal formation in certain blastemata pertaining to the vascular system, in the superincumbent layers upon the inner membrane of arteries, in the fibrinous coagula within the vascular system, in the thrombus, &c.

Within the cancelli small concretions occasionally form, corresponding to the phlebolites of veins.

We have seen these cavernous textures developed in the subcutaneous areolar tissue of the thigh, and communicating with the saphena vein, as also in the substance of the lips. We have seen them as tumours growing out of the diplöe of the skull-bones, and penetrating the compact outer skull-plate, and, again, in the texture of the pia mater. They are the most frequent of all, and the most various in dimensions, up to, and beyond those of a duck's egg, in the liver, where they communicate with branches of the portal vein.

(*b.*) *Fungus hæmatodes, blood fungus.*—What is commonly held to be *fungus hæmatodes*, is a luxuriant vascular growth, mischievous, both from its liability to occasional hemorrhage, and from its tendency to exhaust the constitution by the habitual hemorrhage consequent upon its ulceration. Some deem it curable by removal with the knife, whilst others maintain that it invariably recurs either at the same spot or elsewhere, and that it is of cancerous nature. Some even fancy, that not only does it return in its primitive form, but that it may be replaced by another form of cancer, namely, the medullary or encephaloid.

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[1] The author has since modified his opinions respecting these, so called "cavernous textures." Abandoning the ground of their development out of a solid blastema through partial resorption, he now regards their stroma as nearly identical with that of cancerous structures, both in its elementary constitution, and in the fact of the same dendritic excrescences springing from its septa, and growing into its chamberlets. The affinity of these tumours to cancer, the author considers further established by their not unfrequent concurrence in the same organ (the liver, for instance) with cancerous tumours.

Incipient cavernous tumours do not, it is asserted, present any anastomosis whatever with the venous system. The anastomosis is established only at a later period through the mediation of very minute venous offshoots. It is not made clear, however, in what manner this communication is brought about. (See Rokitansky "über die Entwickelung der Krebsgerüste." Sitzungs-berichte der Kais. Akad. der Wissen schaften, März, 1852.)—Ed.

Our own opinion respecting the occurrence of fungus hæmatodes, is to this effect:

1. It is incontestable that highly developed, bleeding, ulcerating *telangiectases*, as also *cavernous textures*, are frequently mistaken for *fungus hæmatodes*. This applies still more forcibly to a highly vascular, bleeding, ulcerating, blood-turgid medullary carcinoma. Nay, we have often seen a tumour, diagnosed as fungus hæmatodes, resolve itself in the dead subject, after collapse of the blood-vessels had taken place, into a medullary carcinoma.

2. We are convinced of the existence of new-growths almost wholly made up of blood-vessels; we even regard it as likely that the so-termed telangiectases (both congenital and adventitious) are, for the most part, new-growths of blood-vessels.

The question with respect to such luxuriations of blood-vessels is, what is it that determines their (innocent or malignant) character? We believe this to reside, not in the blood-vessel growth, but in the remaining, namely, the intervascular, portion of a common blastema; just as in the malignant osteoid, not the bone-luxuriation, but the adjacent, soft heterologous parenchyma, determines its cancerous nature. Scanty as is the proportion of this inter-vascular heterologous substance to the blood-vessel growth, the former merits, in this sense, the most ample consideration, and however important blood-vessel luxuriation in itself may be, the term fungus hæmatodes designates but a secondary feature of the entire new-growth, a modification, through accidental excess of vascularity, of a new-growth well marked, only imperfectly examined. The designation *vascularized areolar tissue* or *vascularized cancerous formation*, would be both more philosophical and more practical.

Hence the benign or malignant character of a new-growth associated with excessive vascularity would, in such cases, be primitive.

Can the benign new-growth of this kind become malignant? It can hardly be doubted that vascular tumours may accidentally become the nidus of a malignant new-growth, just as may any natural organ rich in blood-vessels.

Another kind of consecutive degradation to malignancy is also conceivable, although for the present little more than hypothetical.

To explain our meaning it will be necessary to enter upon a little further discussion.

Blood-vessel luxuriations represent, when the anastomoses with the old vessels are completed, a new vascular apparatus complete within itself, a repetition, so to speak, of the portal system.

The anastomoses might be of such a character as to attach solely either to the arterial or to the venous system. Van der Kolk considers this proved, maintaining that medullary carcinoma belongs exclusively to the arterial, and another new-growth, which he denominates fungus hæmatodes, to the venous system alone.

A vascular apparatus of this kind not being conceivable without interchange of matter, it might be not unreasonably inferred—

1. That the products of such a process would differ according to the arterial or to the venous nature of the blood circulating in the tumour, and that the products would be more especially anomalous where blood, previously rendered venous, has to permeate a secondary system of capillaries. In both cases the circulation must needs become torpid, and prone to undergo stases.

2. That the crasis of the general circulation must suffer a change, more especially in the second case.

In such wise, vascular luxuriations might indeed be imagined to pass into malignant new-growths, the product, namely, of their interchange of matter constituting a malignant blastema.

The new-formation of *lymphatics* has been demonstrated by Van der Kolk in adventitious membranes and in cancers.

7. *Fat formation, fatty degeneration.*—The anomalous occur-

rence of fat is no less frequent than multiform. The subject is daily acquiring fresh interest, in proportion as the importance of fat in the animal economy, from the incipient stage of digestion through every process of assimilation, up to the formation of the elementary cell, renders itself more and more apparent. It is, however, quite within the compass of pathological anatomy to testify that protein substances, and in particular fibrin and albumen, are capable of undergoing conversion into fat.

A. FAT TEXTURES.

1. *Normal.*—We have already adverted to the excessive formation of fat generally, and to its accidental unequable accumulation about certain organs, amid *general* wasting of this tissue, for example, in the omentum, the kidneys, the mediastina, on the pericardium, and on the heart. We have here to advert more particularly to fat collections still more marked by their locality and limitation. Of this kind are—

a. Those collections of fat important in various ways, which encircle diseased, and especially calculous kidneys, or such as have become atrophied through Bright's disease, or surrounding anomalous bone formations (offering some analogy with the medullary system of bone), as in ossifying arteries, in the vicinity of cancers, &c.

b. *Lipoma* or fatty tumour, an accumulation of adipose tissue in the shape of a spherical, oval, lenticular, more or less lobulated tumour, invested with a delicate capsule of areolar tissue, and permeated by equally delicate and spare continuations of the latter. Its size is from that of a hemp-seed or a pea, to that of a man's head, or more.

It consists, as a mere repetition of the normal adipose tissue, of spherical fat cells, wherein are discoverable stellate or radiating crystals of margarine or margaric acid. Where the areolar tissue capsule is more strongly developed, the lipoma is an *encysted* one. We have met with such encysted portions in the midst of loosely lobulated lipomata.

The lipoma occurs chiefly in the subcutaneous areolar tissue, more especially in localities where fat is deposited in more than ordinary amount in the healthy state, and where accidental circumstances cause its still further accumulation, as at the glutæi, at the thighs, at the back and neck, about the shoulder. It is, however, also observed in parts less abounding in fat, as beneath the hairy scalp. Again, it occurs in the submucous areolar tissue of the stomach, of the intestine, even of the bronchia; in the subserous areolar tissue of the parietal, as well as of the visceral layer, although more commonly of the parietal, in serous and synovial sacs; for example, beneath the pleura and peritonæum, upon the inner surface of the dura mater, and upon the investing membrane of the ventricles. It has been fully described as it occurs in synovial sacs, more especially of the knee-joint, in that peculiar form which Joh. Müller has denominated *lipoma arborescens*; a form to which all lipomatous accumulations beneath serous sacs incline. We have also met with lipomata in glandular organs, more especially in the lungs, liver, and kidneys, in bone affected with osteoporosis and eccentrical atrophy.

Large lipomata in subcutaneous and in submucous areolar tissue, by dint of traction, acquire a pedicle, and lapse into the cavity, it may be of the intestine, in the semblance of a polypus.

Lipomata occur, for the most part, solitarily. Instances are not quite rare, however, of several, or even many coexisting beneath the subcutaneous areolar tissue. Such cases are the more remarkable, that they may affect individuals not otherwise at all prone to excessive fat formation.

In itself lipoma is innocent. By compression, traction, and hampering of space, it may be rendered nocuous. Moreover, its general integument may, through violent tension, through inflammation and suppuration, or through a sloughing process, occasion consecutive ulceration of the tumour, and exhaustion of the powers of life.

It is seldom traceable to palpable mechanical injury. In the majority of cases, more especially where several lipomata concur or succeed each other in growth, there is evidence of neither blow nor compression.

The term *steatoma*, so often misapplied of old, has been reserved by Johannes Müller to designate a peculiar species of lipoma, in which the fat texture is lobulated, as it were, through the intervention of a permeating membrane of areolar tissue, the latter forming a main constituent of the new growth, and imparting to it greater toughness.

2. *Abnormal* (*fat texture.*)—To this category belong, in the first place, cases in which the *contents* of the fat-cells vary from the natural character; in the second place, cases in which the cells themselves deviate from the normal type.

(*a.*) The fat-texture manifests, under certain conditions, anomalies for the most part referable to the nature of the contained fat;

(*α.*) The latter being sometimes preternaturally diffluent, like oil-fat (oleine). It is more than usually unctuous. On pressure or incision it gushes forth abundantly, and in big drops. In the dead subject, neighbouring parts are often found infiltrated with liquid fat. It is mostly tinged of a deep yellow, and resembles marrow.

These characteristics very commonly attach to the fat of the old and cachectic, labouring under osteoporosis (from atrophy), extensive ossification of the arteries, osteomalacia; and of younger individuals a prey to cancer. Similar properties belong in an especial manner to fatty accumulations usurping the place of muscle or of pancreas texture.

(*β.*) The fat contained within the cells is firmer, stearine-like, resembles mutton suet, and dulls the blade of the scalpel. The entire fat of the individual is of this character, more especially that of the subcutaneous adipose tissue, and it is commonly associated with strongly developed pigment formation in the rete mucosum, and a copious secretion from the sebaceous glands,—an oily skin. These properties mark, in an especial degree, the fat of younger dram drinkers, and are almost without exception concurrent with lardaceous affection of the liver.

This variety may be caused by the fat containing a larger proportion of margarine; perhaps, also, by the development of stearine. Spirit-drinking has a very marked tendency to produce it.

(*b.*) The fat-texture gives evidence of anomalies both as to the contents of the fat-cells, and as to the properties of the cell itself.

The *cholesteatoma* of Johannes Müller belongs to this class. It is invariably a local, circumscript new growth. The cholesterine holding stratiform fat-mass consists of thin, sometimes concentrically stratified, mother-of-pearl like, lustrous plates or scales, which, on a closer inspection, appear composed of partly spherical and oval, but for the most part polyedrical vegetable-like cells, one eighth to one sixteenth of a millemeter big. This texture accords with that of the tallowy, adipose tissue of the wether, only that the cells are smaller and more delicate. The majority of the cells do not appear nucleated; many others, however, more especially the younger spherical cells, show distinct nuclei.

Between the layers of this polyedrical cell-texture are visible crystalline deposits of fatty substances, mostly in the shape of rectangular tables. Barruel found them to contain cholesterine and a fat akin to stearine. The cholesteatoma commonly occurs encysted within a fibroid envelope, or within a cyst-membrane lined with a delicate epithelium. We have, in common with other pathologists, seen it thus in the subcutaneous areolar tissue, in bones,—those of the skull in particular,—in the pia mater, and in the brain. Johannes Müller met with it in cysto-sarcoma. It also occurs free, in the shape of a layer, as Cruveilhier has observed in urinary fistulæ, and Johannes Müller and myself upon the surface of an ulcerating mammary cancer. In our own case it was upon the sore surface of a fibrous cancer combined with epithelial cancer. We have met with it in the same combination upon the surface of a sloughing ulcer.

The cholesteatoma is in itself innocent. Inclosed within a cyst it usurps the place of surrounding textures, causing the forcible expansion of the osseous texture in bone, and occasionally perforating the common integument, when subjacent to it, becoming destroyed, and thrown off.

3. *Free Fats.*—The occurrence of free fat,—a condition, we think, properly meriting the term of fatty disease—takes place under different circumstances:

1. It is immediately secreted as such. The seat of its deposition are normal textures and their elementary parts, or else pathological growths.

To the former category belong:

(*a.*) Tallowy infiltration of the liver, a condition in the one instance resulting from spirit drinking; in the other, being the concomitant of tuberculous disease. In the former case it is often associated with a stearine-like character of the contents of the adipose tissue.

(*b.*) To the other mode of occurrence belong more especially the fatty contents of encysted tumours.

These fats differ in character. Thus, in ordinary fatty liver it is a normal fat, for the most part rich in elain,—in some varieties of fatty liver, the waxy liver, a more consistent fat, containing stearine and cholesterine.

In various cysts, again, we meet with a fat consisting in different proportions of elain, margaric, stearine-like fat and butyrine. Some cysts contain cholesterine alone; the majority, other ingredients besides In this mode of its occurrence, fat is very generally associated with epidermoidal and with osseous formation.

2. *Fat,* as such, is liberated owing to new combinations taking place amongst the ultimate constituents of a complex formation. Or, again, it is not set free as preexistent fat, but is newly created, and this out of protein substances, by dint of an elementary transforming power.

This transformation or metamorphosis into fat is rendered highly probable, if not certain:

(*a.*) By the large proportion of fat found at various epochs in the place of parts, into whose composition fat assuredly did not originally enter in anything approaching to its subsequent amount.

(*b.*) By this appearance of excess of fat occurring under conditions which preclude its derivation from a vascular system. It takes place in growths destitute of blood-vessels and often remote from vascularized organs; for example, in exsudates, shut up within thick shrivelled sacs unfurnished with blood-vessels,—in stratiform deposits within arteries. It is even very commonly met with at the centre of the said formations, and therefore at the greatest distance from vascularized parts; as, for instance, in the coagula, within the vascular system, in tubercule-granules, and in crude fibre in circumscript masses.

(*c.*) Lastly, by the appearance of fats in the said formations being the forerunner of their entire metamorphosis, and generally speaking of their destruction.

For this adventitious production of fat, more especially as it affects a blastema in the progress of development, or even perfected textures, we find the term *fatty disease* or *degeneration,* peculiarly appropriate; and we consider it to be fraught with quite enough of interest for general pathology, to justify an attempt to enumerate here the various phases of its occurrence.

1. Appearance of fat in the blood [or fibrin] coagula in veins,—the result of blood disease, either spontaneous, or consequent upon infection by inflammatory products.

2. The fatty conversion of coagula developed through similar agency, in the capillary system, as so-called depôts—metastases.

3. In fibrinous and albuminous products of inflammation, —exsudates, and especially pus. This is manifest in the exsudates of serous membranes.

4. In the albuminous, fibrino-albuminous products of Bright's disease of the kidney. The spots involved in the process of fatty conversion are cognizable to the naked eye as dullish white, glistening, for the most part somewhat turgescent points.

5. In lardaceous infiltration of the liver. The lard-like blastema is seen here and there opaque,—of a dull white or whitish-yellow.

6. In tubercle, in incipient softening of the latter, and in like manner in crude fibrinous deposits.

7. In colloid, as met with frequently in the thyroid gland. In colloid of the more consistent kind, it is discernible with the naked eye as opaque, dull-white or whitish-yellow spots.

8. In cancers, where it enters into emulsion-like combinations with albumen, as also into saponaceous, glutinous conjunctions with bases—saponification of cancers. The points de départ are here the yellow, fibrinous masses which either traverse the cancer as a co-called reticulum, or else occur as circumscript accumulations.

9. In atheromatous disease, in the strata that form upon the inner surface of arteries, and in the soft matrix of phlebolites.

10. In the fibroid blastema and texture, more especially of fibroid tumours and exsudates.

11. In the annulo-fibrous tunic of arteries, where it occurs,

either pure or combined with, and dependent upon, stratiform deposits and their metamorphoses—atheroma and ossification.

12. In the muscles, especially the involuntary, and the heart in particular. Here we encounter an obvious conversion of the muscle-fibrils to molecular fat, with loss of the transverse striæ and inflation of the sheaths.

Further investigations are necessary to determine whether milky blood—the peculiar aspect of which is due to fat; and whether the pellet-like excretion of fat from the intestine, should be classed along with the above.

This conversion into fat, affects, as we have seen, now crude fluid and solid blastemata, now such as have attained to various grades of textural development; finally perfected textures.

The form or type under which the conversion takes place is very frequent, more especially in fluid blastemata. It has been described under the head of "metamorphoses of blastemata."

This process is in one sense to be regarded as a propitious event, as reducing certain growths to a condition readier for resorption and for reassimilation. Moreover, it determines a state of involution, isolation, extinction of the involved growths.

In this latter property, it is often allied with ossification and cretefaction of blastemata and textures,—processes offering many points of analogy, and even of affinity with fatty disease from the disengagement of pre-existent fat. We need only advert to the cretefaction of crude blastemata in the coagula of the larger blood-vessels, and in the capillary system, in exsudates, in pus, in tubercle,—to the ossification and cretefaction of stratiform deposits in the arteries, of the fibroid textures, &c.,—and compare this process with the collateral one of fatty conversion.

Fats, the product of conversion, may present much variety of character. In most instances they are fluid fats in a state of minute molecular subdivision, in larger, lustrous, strongly refracting,—or else, in less bright, yellowish, tough globules. In exsudates, in tubercle, in colloid substances, in cancers, and especially in the atheroma of arteries, cholesterine is frequently encountered in a crystalline state.

(8.) *Epidermidal and Hair formations*—The excessive production over expansive surfaces, both external and internal, of epidermis, with a normal form and aggregation of its elements, is often well exemplified, so far as the mucous membranes are concerned, in those of the œsophagus and vagina. There are, however, epidermidal luxuriations besides, marked by several peculiarities, such as site, circumscribed locality, unusual aggregation of elements. To these belong, also, the epithelial layers investing the various cysts.

(*a.*) As *epithelial* contents of encysted tumours. The form of the cells is most commonly that of tesselated epithelium cells.

Upon the external skin these tumours manifest themselves as luxuriant new-growths, sometimes overspreading a wide surface, sometimes limited to a smaller space, occasionally as cyst-like developments of cutaneous follicles with their excretory ducts. These growths not rarely attain to a considerable circumference, and are distinguished by a peculiar anomalous arrangement of their elements, as also by, on the one side a retarded, on the other an excessive, horny character of the elementary cells.

(*b.*) *Clavus*, a local accumulation of epidermis-cells, of a conical shape with the apex pointing to the interior of the papillary body, with a superimposed disposition of the cells not deviating from the normal.

(*c.*) *Warts.*—Of these there are sundry varieties. The most ordinary consist of cornified epidermis forming a sheath-like receptacle of considerable thickness for the hypertrophied cutaneou papillæ. Others are marked by the elongated fibrous arrangement of very luxuriating cells, as polyedrical, edged cylinders in parallel array, some of which show imperfect cornification. They have a fibro-villous appearance, are humid, and readily broken up by pressure into fibres and their elements. Their cells are devoid of nuclei, and in only a few instances cornified.

(*d.*) *Ichthyosis.*—The higher grades alone concern us here, the epidermis covering a papillary body, proportionately hypertrophied, luxuriates into polyedrical tessellæ, cylinders, and discs. The disposition of the cells, at least in the cylinder form, is a fibrous one, parallel to its length. The degree of cornification is not in every case the same.

(*e.*) *Horn*—cornu cutaneum—a very common, for the most part dingy-brown, longitudinally ribbed, more or less curved, cylindrical or conical horn-growth, springing from a cutaneous follicle of cyst-like development. It attains now and then to several inches in length. Its structure is seemingly fibrous. The cornification of the cells is very marked. It affects parts abounding in follicles, or hairy surfaces and their vicinity; for example, the forehead, the neighbourhood of the pubes, and again, the back and the upper extremities.

All these growths are in their nature innocent.

Besides these, however, there occur upon the common integuments, as also upon the mucous membranes, growths which, although often extirpated with a favorable result, occasionally prove malignant and assimilate in all respects to cancer. Their elementary cells repeat the form of the non-cornified, nucleated, tesselated epithelium cell, not rarely with a fibre-like prolongation, and whose secondary arrangement often displays the areolar type, or else represents in fibrils a moist velvety growth, similar to hypertrophied cutaneous papillæ.

Anomalous hair occurs in various shapes with reference to the form, colour, length, and thickness of the hair-cylinder.

Besides their appearance at unusual points of the external integuments, especially upon pigment nævi, we have to advert to—

(*a.*) Hair contained within encysted tumours. This is commonly mingled with fat and epithelium. It is extremely frequent in the fatty cysts of the ovaries, but is also found in those of the omentum, of the cutis, of the subcutaneous areolar tissue, and even of the lungs. In these cysts it is often found to pervade the fat, as with a felt growing out of variously-sized patches, closely resembling the cutaneous texture, from the inner surface of the sac. Its development is here seen to be entirely identical with that of hair upon the common integuments.

(*b.*) Hair upon mucous membranes. It has been detected upon various mucous membranes, including even the conjunctiva of the eye.

Very small, partly microscopical, hairs are sometimes mixed up with the contents of encysted tumours,—with cholesteatoma. Those said to occur in the different secretions, the urine for example, are evidently derived from a mucous membrane.

9. PIGMENT FORMATION.

Irrespectively of all other anomalous coloration, but with a retrospect to that conversion of blood-pigment alluded to under the head of *hemorrhage*, we shall here treat of *granular pigment*. It appears under the various shades of jet, of Indian-ink black, of russet, or of a yellow-brown.

It occurs both free and inclosed within cells, in the shape of very small, spheroidal, scattered or clustered granules, together with rod-like molecules (with molecular motion), or else in the shape of larger spherical or spheroidal, in like manner either scattered or intimately grouped and blended corpuscles. The cells are for the most part spherical, but in certain conditions as in cancer melanodes, spindle-shaped, caudate, rarely twigged.

These various modes of occurrence, together with the several shades of the pigment enumerated, are, as we shall afterwards see, partly phases of development of the pigment, partly due to external conditions.

It hardly ever occurs quite pure between the elementary parts of a texture. Generally speaking, it adheres to a blastema at some period of textural development, for instance to inflammatory products, adventitious membranes, colloid, malignant growths (melanosis).

When small in quantity and equably distributed, it determines slate-gray coloration of the textures, or a speckling or streaking with black dots. It may, however, manifest itself in larger knotty accumulations so as to consolidate and lay waste the textures, as for example in the lung.

It affects the fluids also.

Before speaking of its origin, it is requisite to pass in review, *as a simple series of facts*, the several cases of its occurrence.

In normal textures, the sequence of its amount and frequency is nearly as follows:

1. *In the lungs*, its seat is the true pulmonary texture, but also the interlobular areolar tissue. When inconsiderable in quantity, it lightly marbles the parenchyma. When abundant, it forms greater, branched accumulations, and along with these, distinct solitary masses of from a hempseed to a bean in size, and presenting a cut surface of dull metallic lustre. Or, again

circumscribed patches of the parenchyma, especially at the apex, may be so replete with it as to display it as a coherent, black, hardened, impervious mass. It is particularly redundant around shrivelling, cretefying tubercles, healing cavities and cicatrices at the apices of the lung. It is for the most part found pure, free, in the shape of a minute molecule, unattached to any ostensible blastema, and certainly very seldom contained within cells.

In manhood and old age it is but a physiological product. In the earlier periods of life alone, as those of boyhood and youth, or when by its quantity it proves destructive to the pulmonary texture, is it to be regarded as a morbid phenomenon. The notion of its being peculiar to old age is correct only in so far as, in the aged, it is for explicable reasons hardly ever absent. It does not, however, by any means belong exclusively to this period of life. If it almost invariably abounds in the old, it is because in them there has been ample time for its accumulation. Still it is undeniable that the condition of the blood in advanced age, with its augmented proportion of blood-corpuscles, and its simultaneous diminution of fibrin, must essentially favour the deposition of this substance.

2. *In the bronchial glands*, from a speckling and extensive marbling up to the point of considerable increase of volume in the gland, and its conversion into a hard Indian ink coloured tumour, in which the glandular parenchyma has perished. Its amount is here commonly proportionate to that in the lungs. Its form is that of free molecule, very rarely of molecule contained within cells.

3. *Gastric and intestinal mucous membrane.*—In the shape of free molecules, often coherent in larger masses. It occurs thus both in the muco-membranous texture itself, more particularly at the great concourse of the solitary and of the aggregate glands, as also in the intestinal villi, imparting to the mucous membrane an aspect, to the naked eye, of being lightly brushed over with black, or uniformly tinged of a slate-gray. Where the accumulation of pigment is considerable, the part appears of a blackish-gray, or it may be of a deep black.

In rarer instances, the tracheal and bronchial mucous membrane is pigmented—that of the uterus frequently.

In the majority of cases it accompanies the more intense chronic catarrhs (blennorrhœæ) of the stomach and intestines. When affecting the intestinal glands, it points to antecedent hyperæmia, stasis, and exsudation—typhus, for example,—at every age, even in delicate children, to a diarrhœa-like process in the follicles of the colon.

4. *In the mesenteric glands,* it is for the most part limited in amount, and concurrent with pigment in the intestinal mucous membrane. Here, again, it is a sequel to typhous hyperæmia and effusion.

In other lymphatic glands, the seat of hyperæmia, hemorrhage, inflammatory stasis, and exsudation, it is less frequent.

5. In the *central ganglia* of the *abdominal sympathetic,* more especially the *ganglion* of the *solar plexus,* usually combined with wasting thereof, as a sequel to typhous hyperæmia. For the most part small in quantity, has a uniform, pale, slate-gray coloration, or is visible as blackish dots or striæ.

6. In the *common integument,* as the so-called *melasma* of the old or cachectic, as a diffuse suffusion of the common integument with pigment, in the lower extremities, and as knotty pigment accumulations in the face.

In new-growths :

1. In the blood-coagula in arteries, veins, and capillaries (metastases), whether spontaneous or due to inflammation of their coats, and terminating in their transmutation to fibroid shrivelling cords and cicatrices.

2. In *atheroma* that has discharged itself into the canals of arteries, and especially in the depôts and cicatrices formed in the act of its excretion.

3. In the *membranaceous growths investing hemorrhagic depôts,* as also in the *contents of hemorrhagic cysts,* being here of a russet or yeast-colour. The black pigment found in the shrivelled and extinct ovarian follicles, after elimination of their contents (during menstruation), is here deserving of mention.

4. In *inflammatory products* upon serous membranes, as a black pigment, upon the peritonæum, more commonly of a brown, or rust-colour upon the tunica vaginalis testis, upon the pleura, the pericardium, the arachnoid. It adheres to the exsudate from the commencement, that is, from its crude condition, through all its phases of textural development, up to the areolar or the fibroid structure. According to its proportion,

it manifests itself as spotted, striated, or uniformly slate-gray, blueish-black coloration,—always occurring as free pigment molecule.

It is more rare in the inflammatory products of parenchymata. Scar-textures are, however, not exempt from it, even in the common integuments.

To its appearance on serous membranes we have to add that detected upon the inner membrane of cysts, and of the cyst-like developments of various hollow organs and canals—for instance, upon the inner surface of the dropsical Fallopian tube sac.

5. In *tubercle,* that is to say, the hemorrhagic tubercle, both in parenchymata and upon serous membranes.

6. In colloid,—mostly as a brown tint.

7. In *cancer melanodes* (commonly called *melanosis, malignant melanôsis*), a heterologous growth, consisting of medullary carcinoma with pigment. The brown and black pigment is here partly free, partly contained in cells, with the character of cancer cells. The medullary carcinoma is in various degrees spotted or striated with the pigment, or, in fine, so replete with it as to appear throughout dark brown or black. (*See Cancer melanodes.*)

In *fluids:*

1. In the fluid portion of the exsudate in serous sacs.

2. Mingled with the contents of the larger cysts, and of hollow organs in process of cyst-like development; for example, the dropsical tube-sac.

Finally, it occurs under several *special conditions,* as:

1. In the black substance present in acute softening of the stomach; in the black contents of the stomach and intestines generally.

2. In the pulp constituting the rare black softening of the spleen.

3. In the detritus of necrosed textures, especially in dry gangrene, or mummification.

4. In the parietes of ill-conditioned abscesses—ichor depôts; but most of all at the margin and base of every variety of intestinal ulcer.

This preliminary will serve as a useful starting point for an inquiry as to the groundwork of pigment, and the conditions upon which its appearance depends.

That the ground-work of pigment is the colouring matter of the blood appears to us proved, the cases in which pigment is obviously derived from hæmatin and blood-corpuscles being so numerous as to exclude all doubts on the subject. Still the circumstances by which the conversion is brought about, and still more the various shadings of the pigment are unexplained.

The cases in which the metamorphosis of blood-pigment—that is of blood-corpuscles into pigment—is manifest, are of the most common occurrence. Such are the cases of hemorrhage, and of hemorrhagic exsudates in serous sacs, more particularly the peritoneum, of hemorrhage from intestinal ulcers, of pigment development in blood-coagula within vessels, of black or dark-coloured softening of the stomach, of black contents of the stomach and intestines generally, &c.

But hemorrhage, whether simple or combined with inflammatory exsudation, cannot, in all instances, be assumed, still less proved. In the other cases, therefore, where pigment occurs, for example, in the lungs, the lymphatics, &c., we must, whilst still holding on to the belief that hæmatin furnishes the ground-work of the die, look around us for some further mode of elucidating the mystery.

The pigment may, independently of any development out of extravasated blood, be brought about through—

1. The obliteration of small blood-vessels or capillaries, with the conversion to pigment of their contained blood, just as the stain is produced in plugging blood-clots within the greater vessels. As the vessel's parietes disappear through absorption, striated accumulations of pigment corresponding to the course of the vessel, are entailed in the textures. This takes place more particularly in membranaceous areolar tissue formed upon serous membranes previously vascularized. In these the opportunity sometimes offers of tracing the aforesaid process. Even the pigment in callosities entailed by so-called capillary phlebitis may be partly brought about by the same means, namely, by metamorphosis of the blood-coagulum in the vessels destroyed.

2. Through conversion to pigment of the blood in different blastemata, especially the products of inflammation and cancer melanodes.

3. By transformation of the blood-pigment along with other

substances in the normal act of nutrition, or in consequence of hyperæmia and inflammation. The probability of this event will be relative to the predominance of the blood-corpuscles in the circulation generally, to the number of old and very highly coloured globules present, and lastly, to the extent to which their colouring matter is taken up by the plasma when attenuated through the diminution of its salts, or the destruction of its fibrin. In this manner it is intelligible how pigment may become engendered without the extravasation of blood-corpuscles, how it so often becomes deposited in the lungs, as the central receptacles for venous blood, how the aged are peculiarly prone to its deposition, and lastly, how in certain crases, the typhous for example, it so frequently attaches as the residue of hyperæmia and inflammation, to the follicular apparatus and the mesenteric glands.

It would seem to have arrived at certain organs partly through resorption—the bronchial glands, for instance.

Our own investigations concerning the morphological process of pigment formation have led to the following conclusions:

The pigment differs according as its basis consists of hæmatin alone in a state of solution, or of blood-corpuscles.

In effusions reddened by dissolved hæmatin, the pigment separates as a result both of the changes produced by resorption, by the accompanying menstrua, and by consolidation, and also of probable external agencies effecting coagulation or precipitation in the shape of a granular mass (of discrete or agglomerated molecular granules), which imparts a brown, a yeasty, or black coloration.

Where *blood-corpuscles are actually present*, either these become dissolved, and the development of pigment out of the colouring matter takes place as in the foregoing case; or else the hæmatin becomes pigment within the blood-corpuscles, which thereby become transformed into mulberry-shaped corpuscles. These remain separate, or cohere in groups of two, three, or four, or they may even gather together into a lobulated mass. Earlier or later they break up into the molecular pigment-granules before adverted to. Under both forms and modes of development the pigment is, to a greater or less extent, contained in cells. Upon this point, experience has shown us that—

1. Preexistent nucleated cells (of various forms) take up hæmatin, which, as the contents of the cells, becomes molecular pigment. This is, perhaps, a repetition of the process that takes place in normal pigment formation.

2. One or more mutually coherent blood-corpuscles constitute, as it were, a nucleus-formation, around which a cell-wall developes itself. Even within this cell the nucleus-mass may break up into molecular pigment. The hæmatin frequently associates itself, dissolved, to the cell's contents, and there coagulates to molecular pigment, whilst the now colourless nucleus-mass (blood-globules) undergoes, probably in its protein contents (globulin), conversion into fat-globules.

3. A cell-wall forms around a conglomeration of molecular granules.

These are frequently all concurrent processes, just as happens with pigment formation external to cells. The two former processes are, however, both attested in coloured exsudates, and most particularly in *cancer melanodes*. In either way, a sort of pigment granule-cell is brought about.

The precise manner in which the conversion of hæmatin to pigment takes place, is obscure, if not altogether unknown. It cannot be doubted that the hæmatin undergoes various and considerable changes. Some indications in point are seemingly derived from the conversion of hæmatin into pigment, through the palpable influence of chemical agents addressed, sometimes to the hæmatin itself, sometimes to the iron it contains. Strong mineral acids (sulphuric, for instance) introduced from without darken or blacken the blood with which they come in contact. Carbonic acid gas (evolved, along with carbonic oxyde gas, out of glowing charcoal) acts in the same manner upon the capillaries when a stream of it traverses the fauces; and a similar influence is exercised by acid secretions generated in the organism itself, as we have seen in alluding to coloured softening of the stomach.

Like the blood itself, the kindred spleen pulp (spleen corpuscles) suffers the same transformation of its elements. The change of colour is here most probably determined through the combination of hæmatin with different acids, carburet, chloride of hæmatin, &c.

The very frequent conversion of red hemorrhagic exsudates upon the peritoneum into black strata, is most probably founded upon the influence of the intestinal gases upon the hæmatin. In common with ammoniacal gas, it is principally the sulphuretted hydrogen of the bowel which, acting (by exosmosis) upon the iron of the hæmatin, enters with it into a black combination, namely, sulphuret of iron. A similar effect is wrought by phosphuretted hydrogen in abscess and gangrene.

Fertile in results as are the above anatomical data relative to the fundamental principle of pigment, they seem to throw very little light upon the chemical processes by which the conversion of hæmatin is regulated. The influences adverted to under which hæmatin blackens, admit of no ulterior application. We are still reduced to the entailed general view of defective decarbonization of the blood, to which the abundance of carbon detected by analysis in the various black substances, certainly adds weight.

But even should the pigment, as Guillot affirms, of the black pulmonary artery, consist of pure carbon, this would in nowise refute our theory, namely, that it is invariably developed out of hæmatin.

Although russet- and yeast-coloured pigment are obviously derived from the same uniform base with black pigment, yet the conditions upon which the existence of this pigment depends are little known, and its composition still less. Thus much is certain, namely, that in colour it is susceptible both of deepening into blackness, and of fading into paleness.

Generally speaking, an organ is liable to become the seat of pigment formation proportionately to its vascularity, to its proneness to hyperæmia, inflammation, and hemorrhage, and to the extent to which its blood-supply is marked by excess of colouring matter, that is, by the venous character (Venosität).

The resorption of granular pigment is a fact! How this takes place,—how and whereby it becomes adapted for the process, is not known.

In itself pigment is an innocent new-growth.

It is still of some importance to inquire what is to be thought of the distinction of pigment into *true* and *false melanosis*. Seeing that pigment has, under all circumstances, one and the same fundamental principle (hæmatin), and that our knowledge of its workings is limited, the distinction seems supererogatory. Which *is* the true and which the false?

We deem it most advisable to abolish the word *melanosis* altogether, and to substitute for it the term *pigment,* designating all growths, normal or pathological, into whose composition pigment enters, as *pigment-holding* or *pigmental,* and what has been called malignant melanosis as *pigmental cancer*, or cancer melanodes.

COLLOID.

Colloid, colloid substance, is a sufficiently common heterologous formation. It is requisite, however, to state, that under this term substances have been brought together which, in a physical and chemical respect, are not perfectly uniform; for instance, the colloid of the thyroid gland, the substance of collonema, on the one side, and on the other, gelatinous cancer. Further inquiry may, however, show such differences to be but modifications and gradations of the same substance.

Moreover, the occurrence of colloid abuts so closely upon the physiological, that it is difficult to define its pathological significance. Thus, it accompanies the often mere passing development of the thyroid gland, the secretion of certain follicles undergoing occasional cyst-like development, especially at the cervix uteri; and again it forms the contents of glandular growths in the progress of cyst-like dilatation. In other instances the appearance of colloid is too obviously of pathological import to admit of any doubt. It constitutes both innocent and malignant new-growths.

Colloid is a semi-fluid adhesive substance, resembling a saturated solution of gum or glue, or a fruit jelly. It is seldom colourless, ordinarily of a honey or pale wine colour, but often brown, or green, sometimes black. With all these tints it is clear and pellucid, and only now and then turbid, flocculent. Microscopically examined, it displays, in smaller or greater number, elementary granules, nucleated forms, nucleated and non-nucleated cells, together with parent cells, in rare instances (even in colloid of the thyroid gland) the *pouch-like* formations mentioned under the head of metamorphosis of blastemata, and even caudate cells.

With respect to chemical composition, the reactions are those of various gradations of casein, of pyin, of certain kinds of mucus.

Colloid is, for the most part, found accumulated in hollow organs, in follicular, alveolar, cystoid spaces, and so seldom free within a texture, that the former mode of its occurrence has been regarded as pathognomonic of its true character.

1. It is most frequently met with in the *thyroid gland*, so frequently, indeed, that few thyroid glands are examined in which more or less of it is not here and there detected. It is accumulated in the cyst-like dilatations of already existing acini, as well as in others of new-growth. The disease represents lymphatic, and in further development, cystic goître. It occurs, moreover—

2. In *simple cysts* (whether new-growths or morbid developments of pre-existent hollow organs, for example, cysts of the kidneys consecutive to Bright's disease), and also in compound cystoids,—of the ovary, for instance.

3. In the *pituitary gland*, as a pale amber-coloured layer interposed between its two lobes, believed by Wenzel to be the cause of epilepsy.

4. In *serous sacs*, as a remarkable transformation of a fibro-croupous exsudate into colloid. Andral has witnessed this in a pleural, and we ourselves in peritoneal exsudates.

5. A colloid substance constitutes *collonema*, and its kindred, benign, new-growths. (See Sarcoma.)

6. A colloid resembling the vitreous secretion of mucous follicles forms the contents of alveoli, and of their endogenous cysts, in innocent and malignant new-growths,—sarcomata and cancers, especially true alveolar cancer.

A question of great interest is, whether *colloid* is secreted as such.

(*a*.) Several facts, especially the appearance of colloid in the Malpighian bodies of the kidneys, but likewise the transformation of the aforesaid exsudates into colloid, afford conclusive evidence that, under some unascertained conditions, albumen and fibrin become converted into colloid.

(*b*.) Other facts render it probable that it is the product of an altered function of secreting gland-cells, or of the action of anomalous cells, parent-cells, alveoli, and cysts.

(*c.*) The colloid of the thyroid gland in its voluminous occurrence, as endemic goître, merits an attentive consideration on account of its character of exclusiveness in relation to tuberculosis. The alienated habit of body acquired in endemic goître, may, indeed, be indicative of a change in the crasis; although as to the nature of such change, its relation to the function of the thyroid gland, and its character of antagonism with tuberculosis, we are altogether in the dark.

Colloid undergoes many spontaneous changes. Besides its resorption, as observed in colloid of the thyroid gland, it becomes, in cyst, diluted by the thin secretion from the cyst wall, or else, under gradual extinction of the cyst, condensed, and eventually changed into a brittle substance resembling dried glue. Lastly, it achieves—

(*a.*) A remarkable conversion to molecular fat, becoming yellowish, turbid, opaque, and unctuous (colloid of the thyroid gland, gelatin of alveolar cancer).

(*b.*) In a few instances, cretefaction,—in colloid of the thyroid gland. These cases are, however, quite distinct from the cretefaction and ossification of fibrinous exsudates within the strumous thyroid gland.

CYST AND ALVEOLUS.

In asserting cyst to be a substantive new-growth, with a distinctive elementary groundwork, we exclude all accidental cyst formations, that is, capsules and sheaths forming around foreign bodies, extravasate, or entozoa (cysticercus, for example); as also cyst-like disease of hollow organs consequent upon the closing and obliteration of their excretory ducts and orifices,—for example, in the gall-bladder, the Fallopian tube and uterus, the vermiform appendix, the sebaceous and muciporous glands. Certain gland-elements, however, and in particular those of the thyroid gland, demand an especial consideration, inasmuch as these hollow bodies represent a certain stage in the process of cyst formation, having the same elementary structure, and being susceptible of ulterior cyst-like development.

Let us begin with the results of an examination with the naked eye of perfect cysts, and in particular of the exquisite specimens so frequently met with in the ovaries.

We have the simple (unicancellated) and the compound cyst (Müller's compound cystoid). The first is sufficiently characterised by its name. Compound cysts declare themselves by phenomena which induced Hodgkin to distinguish them in two classes, although types of both very commonly coexist in the same formation. The first comprehends a cyst-formation with cysts of a secondary order in the parietes of a voluminous (parent) cyst; and these secondary cysts involve, in like manner, cysts of a tertiary order within their parietes. These filial cysts project upon the outer surface of the parent cyst, rather than upon its inner surface, where they are in a degree flattened. The wall of the parent cyst often appears separated, receiving the secondary cyst, as it were, in a chink.

Such a formation is to be distinguished from a group of simple cysts developed in mutual juxtaposition, some one of which predominating in size, flattens the contiguous smaller ones. A group of smaller cysts in an ovary may readily mislead; making it seem as if the fibrous capsule of the ovary were but the wall of a cyst, and as if the smaller cysts interposed between it and a contiguous larger one, were secondary cysts.

The repetition of this process of secondary cyst-formation frequently leads to a very complex cyst-formation, wherein, however, for the most part, a cyst preeminent in size, reveals itself as the parent or primary cyst in whose parietes the cysts of the second order become developed. Sometimes the primary cyst is so prolific of this secondary cyst development within its parietes, as to endow the latter with a considerable thickness. It may even cause them here and there to degenerate into a tumour, consisting of an aggregate of cysts, collocated like facetted pouches in the breadth of the cyst-wall, and presenting a polyedrical cell-structure, in and upon the walls of which smaller cysts arise. Occasionally a cyst-wall, so constituted, further degenerates (owing to a rupture of the secondary cysts, and to their bursting into the cavity of the parent-cysts) into a multilocular cell or network.

Every cyst is of course competent to represent a parent cyst in relation to its own ulterior cyst production.

The *second* category comprises cysts in which secondary cysts arise upon the internal surface of the parent cyst, and grow into its cavity. They are sessile upon a broad base, or more often upon a neck or pedicle; in which case they mostly represent pear- or wedge-shaped tumours. They often so luxuriate in number, and at the same time grow to such a size as nearly to fill a parent-cyst of considerable magnitude. In rare instances, a solitary cyst of this kind so increases as singly to fill up the space of the parent cyst, causing the sac to consist, down to the base of the filial cyst, of two contiguous layers.

These secondary cysts become developed in the internal layer of the parietes of the parent cyst, and have a sheath derived from the internal membrane, from which the secondary cyst can, with care, be separated. In the pedunculated, pear-shaped cysts, it furnishes, in a state of involution, the pedicle into which the pouch, or wedge-like cyst, projects with a conical tapering end.

They are either simple or compound, according to one or other type. In their wall, namely, reside cysts of an ulterior, that is, a tertiary formation, which grow more or less outwardly or inwardly, the former acquiring a shallow-lobed, blackberry-shape, and appearing cellular within. The pear-shaped cysts commonly consist of several parallel pouches of various lengths. Along with these are found, on the inner surface of the parent cyst, in varying numbers, the smallest vesicles, just cognizable with the naked eye. In one instance these were found on the inner surface of an extensive ovarian cyst, mixed up with, for the most part, naked yellowish incrustations the size of a poppy- or a millet-seed.

The difference between these two types of the compound cyst is obviously not essential, but depends only upon the seat of development of the secondary cyst. Hence, the very common concurrence of the two types.

There also occur, on the inner surface of the cysts, both parent and secondary, ramified cauliflower excrescences, flattened, or fungoid, or pedunculated. These are scattered singly, or grouped together, or knotted in masses. Sometimes they luxuriate in and by the side of the said secondary cysts, to such an extent as to fill both these and the parent cyst, rupturing the latter, and, in the frequent cases of ovarian cyst, invading the peritoneal cavity. In the ruptured secondary cysts we often recognise their sheaths folded back, and reflected over the cauliflower vegetation.

Besides the variations alluded to, there is much that is worthy of note in and about these excrescences.

1. They consist of a very delicate membranaceous growth folded and rolled up in their pedicle, projecting about and especially above it in various ways, branching out into numerous villous and bulb-like processes, or into the semblance of a plaited frill. Upon the said processes are again seated delicate villous flocculi. They are highly vascular, and have a blood-loaded aspect.

2. Here and there they frequently carry, especially at the extremity of their branchlets, a just cognizable, poppy-seed-sized, limpid, or semi-opaque vesicle, or a hemp-seed-, or a pea- or bean-sized cyst.

3. More frequently still they bear upon their twigs solid, though soft, whitish, roundish, or, from mutual compression, indistinctly facetted corpuscles; or else tougher, white, opaque tubercula, mostly of the bigness of millet or hemp-seeds. Here the entire excrescence is commonly white, the small broad-based ones resembling delicate stellar horny warts, whilst the larger and more extensively clustered ones constitute an unyielding tumour, superficially stellate, studded with the aforesaid tubercula upon its peripheral villous structure, permeated throughout its cut surface by fibrous threads (the pedicles). In this tumour, the blood-vessels have become destroyed.

4. Besides the more extensive excrescences, smaller ones are commonly seen, resembling a nap of extremely delicate villi, or of finely pedunculated tubercula.

5. At the same time the internal investment of the cyst has, in expanded parts, often a very finely reticulated aspect; or it reveals very minute fissure-like grooves, not a few of which are surrounded by an elevated ridge-like brink. Out of these is here and there seen to arise a simple or branched excrescence. In other places, the internal layer of the cyst wall is seen raised into a flattened vesicle, which, like the said grooves, often displays minute fissured openings. Internally is sometimes plainly discerned a convoluted mass of bulb-shaped excrescences, or else a very minute network.

With these are associated larger vesicles, rising into pedunculated wedge-shaped pouches, which contain a very delicate cancellated structure, and frequently exhibit roundish or angular chink-like openings, out of which delicate felt-like excrescences occasionally project. The size of the cysts varies greatly from that of a just cognizable vesicle to that of a sac of from 1 to 2 lines in diameter. The compound cysts may, of course, attain to a very considerable magnitude, a notable portion always appertaining to the parent cyst.

The free space of the cysts hitherto described, is commonly occupied by a serous synovia-like, or a thicker glutinous, or glutino-lardaceous, so termed, colloid moisture.

Examined under the microscope, the following additional light is thrown upon the above appearances.

The cyst-wall consists of densely-reticulated areolar tissue, the internal layer constituting an epithelium of cells or nuclei. In large cysts this is, for the most part, absent, and the internal layer generally presents a nucleated, structureless, or striated blastema, at the circumference of which the oval nuclei are in the act of splitting into fibres, in the direction of their long axis. On examining a section of the internal layer of a cyst wall, from a part furnished with vesicles (secondary cysts), we obtain a view like that presented by the cortical substance of a kidney affected with cyst formation, to the consideration of which we shall shortly have to recur. The same nidus often contains concurrently incrustations, in some instances remarkable for their size and figure. On examination, the excrescence appears as a hollow growth consisting of a transparent structureless membrane, studded with round or oval nuclei, often striated, especially at the pedicle, and breaking up into delicate fibrils, with numerous spheroid protuberances. These become developed into pouches, mostly bulb-shaped at the extremity, and by throwing out secondary protuberances and pouches, complete the branchlets and twigs of the excrescence. They may be invested with the epithelium of the cyst wall, or even uninvested. They are furnished with conspicuous blood-vessels, which, running along the protuberances, describe extensive arches and anastomoses, and frequently become the seat of aneurismal dilatations, or the source of hemorrhagic effusion into the cysts. In their interior they contain nuclei in various numbers, and along with these, especially near the blind extremity of the branchlets, growths which turn out to be young cysts.

These young cysts dilate into those spoken of as cognizable with the naked eye.

The minutest excrescences appear as simple, smooth, or tuberous hollow bulbs. The internal layer of a cyst-wall, presenting the reticulated texture described (5), appears, when magnified, in the form of elongated, round, angular, distended meshes, through which the simple, smooth bulbs penetrate as they grow. The cancellated framework, contained within the described vesicles, consists of a hyaline, structureless membrane, studded with nuclei. It has unquestionably arisen out of the fusion of several bulbs.

The excrescence, as described at No. 3, arises through the development of areolar tissue out of a transparent amply nucleated blastema. In its cavity are lodged, sometimes in vast quantities, simple and laminated, semi-opaque, incrusted growths, from the size of an elementary granule to a diameter of $\frac{1}{85}$th of a millemeter, the circumference, most common to incrusted cysts.

The excrescence simultaneously becomes fibrous, and shrivels, with condensation of fibrous parts, into the solid masses above described.

In these observations, two phenomena engross our attention, namely, the development of the secondary cyst and the hollow growths forming upon the internal wall of the cyst. We have made them the subject of an extended investigation, with a view to the solution of the double question as to the nature of elementary germ for cysts, both primary and secondary, and of its ulterior development,—and as to the import of the said hollow growths.

(*a*.) The cysts best adapted for the inquiry, are young, small, clustered cysts, just visible to the naked eye; others being probably present, still smaller, down to the germ itself, out of which they spring. The cysts so frequent in the kidneys, or on the broad ligaments, and on the peritoneum of the tubes and ovaries, furnish ample materials for the purpose.

(1.) In the cortical substance of the kidneys, especially during the decline of Bright's disease, a luxuriating cyst-formation is not uncommon.

In the dimpled depressions upon the surface of atrophied, gibbous kidneys, reside entire nests of parallel-clustered, just discernible, poppy-or millet-seed-sized vesicles, imbedded in a reddish-grey, or whitish nidus. Occasionally the kidney is found altogether degraded into an aggregate of various-sized cysts.

A small portion of such a nidus placed under the microscope, displays, along with the débris of renal texture,—namely, uriniferous tubules and Malpighian tufts, in a state of collapse or involution; the former denuded of their epithelium, and here and there replete with fat-molecules—a multitude of cysts invisible to the naked eye. The more marked have parietes, consisting of fibres beset with elongated oval nuclei, which, more particularly about the inner fibre layers, bend round towards the circumference of the cyst. These cysts are replete with granulated nuclei,—now and then with spherical or polyedrical cells, to which, in some few, is superadded a molecular mass, partially betraying by its brown coloration its character as pigment granules.

In some cases this occupies the centre of the cyst, where the nuclei become indistinct and disappear. In some cysts, the nuclei (or cells) are reduced to an epithelial formation investing the cyst. In others, again, even this is wanting, and the sterile cyst is entirely filled with a clear, or semi-opaque, viscid humour. They are of very various size, from a diameter of $\frac{1}{8}$ to $\frac{1}{50}$ of a millemeter, the former immediately preceding vesicles distinctly cognizable with the naked eye. Conjointly with these, are found cysts, which, with similar contents and parietes, consisting of a structureless transparent membrane, reside in an equally structureless stroma, interspersed with oval nuclei, and in progress of development into a fibrillation about to encircle the cyst.

We further discern, commonly within an aggregate of spherical, nucleus-like bodies, growths of various magnitude, down to that which only just surpasses the dimensions of the nucleus. These growths quite coincide with the aforesaid structureless vesicles. The smallest contain a clear moisture, or are faintly granular. In the larger ones, a central nucleus soon appears, joined by a second, a third, a fourth, and more, so as to fill the equably dilated vesicle. This description *comprises the history of the development of the cyst*, and may, under favorable circumstances, be found exemplified in a single preparation. *It is obviously the nucleus that grows up into the cyst*; which, with reference to endogenous production, either generates brood nuclei or else proves sterile.

Besides the nuclei, there are seen smaller corpuscles of all sizes, from that of the nucleus down to that of the so-called elementary granule, and manifesting, in proportion to their magnitude, more and more of the character of the nucleus. It is, therefore, at once to be stated, that the *nucleus has arisen out of the elementary granule*; and this, through spontaneous germination,—not through the agglomeration of several. Finally, we observe, in the preparation, concentrically stratified bodies, also, of different sizes, and consisting of incapsuled vesicles in varying number. These vesicles are themselves sterile, or the central vesicle may have its space occupied by one or more granulated nuclei. Sometimes it is itself represented by a nucleus. One or more of the external layers contain, in like manner, nuclei, oval in shape, and bent to a parallel with the layer. Again, the layers are in some cases slightly gibbous. Incrustations of these forms are also present,—nay, kidneys sometimes occur, in which the cortical substance, otherwise seemingly sound, is interspersed with them, looking like yellowish, transparent grains of sand.

These are the results to which I have referred, in describing the compound cystoid. A similar result is furnished by the inspection of a group of cysts *in the above-mentioned sexual attachments of the peritoneum.*

2. The renal preparation at first sight so much *resembles the texture of the thyroid gland, and more particularly the goitred thyroid gland*, as to render it impossible to discriminate between the two. Simon has directed attention to this in vol. xxx. of the 'Transactions of the Medical and Chirurgical Society.' Not only is the normal gland-vesicle of the thyroid gland identical with a cyst of corresponding size, but the development of new gland-vesicles in a goître is identical with cyst development, and again the preternatural dilatation of the gland-vesicle—its so-called cyst-like degeneration—identical with a cyst outgrowing its microscopical proportions. Nay, the gland-vesicle betrays in its development the same anomalies

as the cyst in its development as a sterile vesicle, or as a laminated cyst in its degeneration to a colloid sphere, and in its incrustation.

3. The same relations attach to *cyst-formation* in mucous membranes. In those of the stomach, the colon, the uterus, a morbid growth occurs, known by the term cell- or vesicle-polypus. It consists of an aggregation of from millet or hempseed to pea-sized cysts, broad-based, but mostly furnished with a neck, and commonly representing the head-like free extremity of a cylinderical prolongation of the mucous membrane. These cysts are developed in the texture of the mucous membrane, seldom exceed the aforesaid volume, but burst and evacuate their viscid, jelly-like contents upon the surface of the mucous membrane. Their fate, beyond this disruption and violence, I have been unable to ascertain. They probably give place to new ones. We may refer here to those bodies suspended by a pedicle of mucous membrane from the cervix uteri, and known as *ovula Nabothi.* These, though commonly received within the domain of physiology, in reality present a continuous cyst-formation, destroyed from time to time by disruption and evacuation. They occur, in like manner, on the mucous membrane of the renal pelves, and of the ureters.

But I have repeatedly observed millet, hemp, nay, almost pea-sized cysts, in surpassing numbers in the mucous membrane of these urinary conduits. Some of them contained a flaky, inspissated, colloid-moisture. In one instance, my attention was drawn to them, by the presence of little, roundish, naked, colloid pellets, in the urinary bladder.

Just as in physiological textures, so also in pathological parenchymata does cyst formation occur. For example; in the textures of *Sarcoma* and *carcinoma,*—giving rise to the family of *cysto-sarcomata* and *cysto-carcinomata.* Even the so-called *carcinoma alveolare* consists, mainly, in cyst-development.

Cancer-cyst varies in respect to size from the microscopic, to the circumference of the colossal cysts in the compound cystoid. The alveoli of areolar cancer in particular—that is, the small cysts constituting alveolar-cancer,—and especially the peripheral-alveoli, grow into comprehensive cysts. The cancer-cyst contains a sero-albuminous fluid, a jelly-like (colloid) substance, and frequently cancer-parenchyma. It is not a rare thing to find—imbedded in a cancer, or independent, and remote from a heterologous growth, ascertained by its volume to be the primary seat of the cancer-production—tumours, obviously consisting of encysted-cancer parenchyma, or cancer-tubera, which, manifestly enveloped in an often stoutish fibrous capsule, are distinguishable at a glance from other uninvested accumulations of the cancerous substance. Within the encysted parenchyma, again, is sometimes lodged a smaller, filial cyst.

Upon this point and upon the development of the cancer-cyst microscopic inspection throws much light. The appropriate materials for examination are afforded more especially by cancer-masses, which, with or without the presence of voluminous cysts, exhibit to the unassisted eye, minute, limpid vesicles, or else an aciniform, glandular structure.

In such cancer-growths, besides the ordinary nucleated cells, often indeed distinguished by their eccentric forms, we discern:

1. Cells of notable diameter, up to $\frac{1}{30}$th of a millemeter, with a very large nucleus, dilated into a clear vesicle, which approximates to, if not touches, the cell wall. In some of these bloated nuclei, a nucleus corpuscle has become developed into a second central nucleus, which, in its turn also, contains nucleus-corpuscle.

In certain cells we find two of these advanced globular or mutually flattened nuclei, as also several without a nucleus corpuscle, or with one which, in like manner, expands into a nucleus. Other cells contain, along with a swollen nucleus, one or more ordinary granulated or transparent, spherical or oblong nuclei, besides.

2. *Divested nuclei* which, like the cell-included nucleus (cell-nucleus), expand into larger, transparent, structureless vesicles. These—

(*a*.) Remain sterile cysts.

(*b*.) They give birth to numerous secondary nuclei, until the cyst is replete with these. Such cysts often entirely resemble the gland-cyst of the thyroid and supra-renal glands. Of these secondary nuclei, one or more occasionally grow into a vesicle, which remains sterile, or fills with brood nuclei, or presents phenomena about to be described.

(*c.*) *A central nucleus corpuscle appears in the vesicle and expands into a secondary nucleus.*—This nucleus, like the primitive one, dilates into a vesicle in which a nucleus formation of the third order takes place.

Out of the frequent repetition of this process originate growths concentrically laminated, or consisting of a series of endogenous vesicles, which here again are distinguished by their proclivity to incrustration. Sometimes there are developed, in one of the secondary vesicles, more than a single nucleus—for example, two out of each, out of one only of which a laminated formation may become developed. In the latter case, ordinary brood nuclei are generated in the other. The inner vesicle either remains sterile, or a central nucleus, or it may be several nuclei, engross its space. Here, again, there may be laminated structures, springing from extra-centrical nuclei.

All these structures are lodged within a parenchyma (differing in composition, and in the fecundity of its elements), of nuclei, cells, caudate-cells, fibres. These elements are so arranged as to inclose the said structures in a capsular fashion, the cells, and even the nuclei, lengthening into riband-like, caudate cells, and oblong nuclei, with a corresponding incurvation. (*Alveolar textural arrangement.*)

Still, the main condition for the growth of the vesicle is the presence of encircling fibres, and their appropriation to the fabric of a resisting fibrous cyst.

The simultaneous evolution of the brood nuclei of the vesicle causes the cyst to become speedily furnished with a proper parenchyma corresponding with that which surrounds the cyst. In this a filial cyst may become developed.

The structureless vesicles alluded to offer many further points of interest:

(*a.*) There is, frequently, a marked difference in the contents both of the simple vesicles, and of the individual layers of the vesicles successively ingenerated. Thus, some appear clear and colourless, others of a reddish tint; in others again, the contents are denser, pearly, or opaque, lightly granular. Some contain granules in various amount, which show themselves to be fat,—fatty conversion of the nuclei.

(*b.*) Of two intussuscepted vesicles the inner one is sometimes irregularly collapsed, wrinkled, or even pretty regularly indented. This probably results from a consecutive difference of density in the contents of the two, a condensation of the contents of the outer vesicle determining exosmotic effusion of the thinner contents of the inner vesicle.

(*c.*) Intussuscepted vesicles are, for the most part, sterile. Within their layers, however, are frequently impacted oblong, curved nuclei.

(*d.*) The layers are commonly smooth; often, however, gibbous, wavy, and curled.

The development of these cysts out of the nucleus, through growth of the latter, is here demonstrable even in the naked nucleus; by growth of the celled nucleus, however, it is placed beyond all doubt. Here again, the elementary granule is cognizable, as the ultimate, fundamental form; the nucleus being obviously and simply developed by growth out of the so-called nucleolus, or elementary granule.

Difficulties might, however, still arise so long as that theory of cell-formation obtains which assigns to the cell a genesis and an import distinct from those of the nucleus. If there be parent cells, their resemblance with the expanding nuclei, both in form, and often in their relation to chemical agency, might render it no easy task to determine the precise nature of a vesicle, seeing that parent cells and parent nuclei are met with concurrently. In a laminated structure, it would be peculiarly puzzling to have to decide, whether its external contour belonged to a cell, or to a nucleus-wall.

From what has been stated, however, the existence of a cancer-cyst certainly may be inferred, a cyst, namely, developed out of the elements constituting cancerous substance, and productive of cancer elements within itself. Not every cyst, however, concurrent with cancer, is necessarily of a cancerous nature, the malignant growth very possibly wearing but the character of an accidental complication.

In cysto-sarcoma the same relations obtain as to the [primitive] development of the cyst.

B. As regards the *excrescences occurring upon the inner surface of the cyst,* repeated observations have established the following facts:

1. Upon mucous membranes, and especially upon that of the urinary bladder, there occurs a cancerous growth which we have elsewhere termed *villous cancer,* as a structure pertaining to medullary carcinoma, and containing within a villo-membranous vascularized stroma, a medullary (encephaloid) cancer juice. Later investigations have led to results which induce me to reopen this subject.

It consists, as a walnut- or a fist-sized tumour, of a multitude of densely thronged excrescences, which, upon a cord-like, longer or shorter, pedicle unfold into delicate membranes, breaking up into numerous ramifications, and again into more and more tender branchlets, wholly overlaid with delicate villi. Many twigs have a grape-clustered appearance, their villi bearing poppy or millet-seed-like, clear or opaque, white vesicles. Larger cysts reside in the primary cotyledon and ramification. Many excrescences, again, represent hollow, shut, or at their free extremity, wide-mouthed pouches. In one instance, the entire growth consisted of polyedrical, at their free ends for the most part wide-mouthed, pouches, densely beset with villi at the brink of the aperture. The tumour is throughout surcharged with a whitish, creamy, medullary juice. It is frequently a more consistent medulla-like mass that fills up the cavities of the growth, which in this case acquires considerable density, and offers proportionate resistance.

The growth is in general highly vascular, and in its recent state, turgid, of a deep purple tint, and prone to hemorrhage. At the base, from whence the tumour commonly rises with a neck, we have found an extensive sinus of a venous kind, upon the inner surface of which are seen numerous pin-puncture and poppy-seed-sized orifices, leading to blood-vessels, which ascend within the pedicles of the excrescences, and accompany their ramifications.

In the vicinity of, or even remote from, the heterologous structure, are smaller groups, or solitary excrescences. These, when young, are very delicate, so as when under water to resemble a fine nap.

Microscopic Examination.—In the cream- or marrow-like juice that exudes on gentle pressure, are found variously-shaped cells, with one or several, in part turgid, vesicle-like nuclei, along with bare, middle-sized, and larger-sized nuclei, furnished with a considerable nucleus-corpuscle, of which one especially, was found large, and presenting internally a dull secondary nucleus contour. This juice resides in the before-mentioned pouch-like chambers. The excrescences are externally clothed with an epithelial layer.

The membranous structure constituting the excrescence appears as a very delicate, transparent, structureless, here and there striated membrane, overstrewn with oblong nuclei, and breaking forth about the pedicle into slender wavy fibrils. It is invested with a simple layer of granulated nuclei for epithelium, which is, however, frequently wanting.

A *clustered twig* appears as a clavate, hollow structure upon a delicately fibred pedicle, young cysts, as structureless vesicles, occupying the interior of the protuberances. Here are seen two outlines, of which the outer one belongs to the protuberance, the inner one to the young cyst; elongated nuclei course along between the two. The cyst is replete with spherical nucleolated nuclei. A few cysts open towards the pedicle of the terminal bulb in which they are contained.

In some of these sacculi are besides found, in various numbers up to the point of repletion, fat globules, some of largish dimensions. These lend to the cyst the white opaque aspect already referred to.

The larger millet-seed-sized vesicles, visible to the naked eye, contain a colourless, tenacious fluid in which the above-mentioned nuclei float.

A morsel of a membranous expansion of the excrescence appears, when magnified by 50 diameters, distinctly to consist of two layers, and is everywhere, but especially at the summit, overspread with numerous bulb-shaped protuberances, which themselves throw out secondary projections; whilst considerable blood-vessels ascend to all. In the inside are here and there seated groups of fat-globules.

Their bilaminated structure renders it more than probable that the layers, in consequence of the copious production and accumulation of the cancerous elements, separate into the pouches aforesaid, which, for the same reason, give way at this free extremity.

In 1842, a urinary bladder was shown to us by Hodgkin, at Guy's Hospital, upon the inner surface of which were seated numerous largish bulb-shaped cysts, filled with the excrescences referred to.

The extensive development of the cysts in a mucous membrane is in itself very remarkable; whether, with the excrescences, they be of a cancerous nature appears uncertain. Upon the *mucous membrane of the renal pelvis* we have seen, along with young cysts, some solitary, others grouped together in its parenchyma, awl-shaped and bulbous, smooth and villous, red, vascular excrescences. They were seated, in part singly, partly in collected groups, some bearing a just discernible transparent globule at their free extremity, which microscopic examination showed to be a young cyst.

2. Upon *serous membranes*, and the peritoneum in particular, we have, in connection with luxuriating medullary cystocarcinoma of the ovaries, met with medullary vegetations which, judging from their appearance and the arrangement of their vessels, would seem to belong to this category.

On the other hand, an examination of the preparations in the pathological museum of Vienna has taught us that the dentritic vegetations which often luxuriate upon synovial membranes, and in the capsule of the knee-joint so numerously that it appears invested with them as with a felt, really appertain to this class. In the interior of the hollow growths which constitute them, a development of areolar tissue takes place, just as in the cysts of the vascular plexus of which we shall hereafter speak, until they at length become replete with it. The largish terminal bulbs of their stems and branches are frequently so flattened as to resemble linseed or melon seeds (Majo).

The entire excrescences with these numerously seated upon them, and it may be in imbricated order, acquire the aspect of foliage. In one instance we found them to contain fat-cells, which explains what Johannes Müller meant by *lipoma arborescens*. In fine, we doubt not that they offer the original nidus for the production of those circular, smooth, or knotted, facetted cartilage or bone plates, which occupy the inner surfaces of synovial sacs, often attain to a considerable volume, and, by spontaneous detachment, become free bodies within the capsule.

3. The encysted parenchyma is often contained free within

the cyst space; sometimes, however, there is present a meshwork, issuing from the inner wall of the cyst, the spaces of which are filled up with the medullary mass. This framework consists of a transparent striated blastema, pervaded by numerous spherical and oblong nuclei, and breaking up into fibrils. Simple hollow bulbs shoot up from its trellises, and into its chamberlets. Respecting the development of this stroma, two theories might be propounded:

(*a.*) It might result from a continuous separation of the internal layer of the cyst-wall.

(*b.*) It is, however, far more probable that, like the framework of alveolar gelatinous cancer, or of the encysted new growth of thyroid gland parenchyma in goitre, it results from the blending of the excrescences concentrically growing from the inner surface of the cyst.

Occasionally, sharply-defined, spheroid cancer tumours occur in which no cyst-wall is discoverable, but yet, in their interior, a stroma of this kind. It is very probable that these tumours were previously encysted cancer-masses, from which the cyst has, owing to a total disruption with excessive production of those excrescences, disappeared or become part and parcel of the stroma.

The repeated examination of so-called *alveolar cancer* (gelatine cancer in alveolar form) offers very interesting results, corroborative of the endogenous multiplication of its cysts. Besides the exogenous augmentation, there occurs likewise an endogenous one, the medium of which is offered by the often-mentioned excrescences; and in this process these become converted into the framework, in the alveoli of which the small cysts are subsequently lodged. From the inner surface of a thick-walled follicle, or alveolus, numerous simple bulbous pouches shoot up into it, so as to penetrate it. Another, contiguous, is replete with a young delicate alveolar parenchyma. In the interior of those hollow bulbs is seen one, or a pair of cysts. In such prolific cysts the development of the fibre-layer for the young cysts may often be seen proceeding from the base of the excrescences, near the inner wall of the parent follicle. Gluge has seen these bulbous pouches in a cancer of the rectum, and figured them. Owing, however, to his treatment of the preparation, he recognised neither their relation to the alveoli nor their character in general. He regards them as altered and hypertrophied muciparous glands.

Thus, encysted alveolar cancer is accounted for and explained in the same manner as medullary carcinoma.

4. Upon the internal parietes of *cysto-sarcomata* there are known to occur (in the so-called *cysto-sarcoma proliferum*) warty, foliaceous, bulbous vegetations, as also pedunculate cysts. The former often attain to a considerable size, so as to fill up the space of the cyst, presenting a flesh-like aspect. [For an explanation of these phenomena, the reader is referred to an appendix to the separate section on *cysto-sarcoma.*]

5. In goître, besides others, there occur cysts filled with thyroid gland parenchyma of accessory growth. We have often the opportunity of witnessing the process of this endogenous production in its incipient stage. From the inner parietes of the cyst arise delicate, transparent, protuberant, vascularized excrescences, in the interior of which a new creation of gland-vesicles takes place.

6. Finally, in the domain of physiology, we encounter, upon the vascular plexuses in the lateral ventricles of the brain, a formation which, as comparative microscopical investigations teach us, fully coincides with the excrescences still before us. Upon the lateral plexuses of vessels are seated great multitudes of delicate, red, vascularized villi. These consist, beneath an epithelial covering, of a transparent, multifariously projecting, fretted, hollow growth, in the protuberances of which run arched blood-vessels of no inconsiderable size. Here we seldom fail, especially upon subjects advanced in years, to find numerous little cystlets, for the most part lodged in those protuberances. Generally speaking they do not outgrow a certain measure, a diameter of from $\frac{1}{25}$th of a millemeter up to a cyst discernible by the naked eye, and differing from what are usually called cysts of the vascular plexus. By dint of a repeated development of central nuclei, the majority are wrought into concentrically laminated growths, and undergo incrustation.

The very common so-called *cysts of the vascular plexus* are not genuine cysts. Numerous examinations have convinced us of their being dilatations of the hollow growth which constitutes the villus of the blood-vessel plexus. This dilatation preeminently affects the villi adjacent to the tortuous blood-vessels upon the convexity of the arch described by the vascular plexus. Accordingly, they are clusters of gibbous and indented vesicles, separated by the indentations into several loculi. Little remnants of villi are often to be detected upon them. We have frequently observed the dilatation of the villus at its commencement. In this manner, the so-called cysts of the vascular plexus answer to the pouches into which the excrescences constituting villous cancer widen. Besides other matters, they contain areolar tissue, sometimes to perfect repletion, so that, in this respect, they may be likened to the excrescences upon the internal membrane of the cysts, and especially to those which, through the development of areolar tissue, have become internally parenchymatous; as, for instance, in the cyst of sarcoma.

The contents of what are called *cysts of the vascular plexus* answer, in all essential points, to the contents of cysts proper. They consist in a watery albuminous fluid, which, with a certain amount and quality of its accompanying organic elements, becomes turbid, thickish, whey-like, and in great measure supplanted by the development of areolar tissue.

These elements are—

(*a.*) Minute, $\frac{1}{800}$ millemeter-sized, elementary granules, free, or numerously held together by a viscid interstitial substance.

(*b.*) Larger, up to nucleus-sized, spheroid, occasionally somewhat oblong vesicles, which speedily increase to $\frac{1}{25}$th of a millemeter. There are, besides, ordinary, granular, spherical, and oblong nuclei and granules.

(*c.*) The larger vesicles are simple, or successively inshrined. Some exhibit a double outline, that of the inner vesicle often lying so close to the outer one as to be easily overlooked. In other cases, the two are widely parted, the inner one being slightly wrinkled or curled. In others, again, there is, between the two contours, at some one spot, a space defined by a single outline, which sometimes resembles a shallow section of a sphere, and is particularly marked where the compressed contents of the external cyst are granular, and therefore contrast with the limpid contents of the inner vesicle. In some instances the inner vesicle is small, and either central to the outer one, or resting upon its wall. The latter kind probably engenders the form just adverted to.

Other vesicles, again, consist of three or of four, the inner, secondary and tertiary ones being by turns central and extra-centrical. This species of complex and involved sheathing is met with in very small ($\frac{1}{266}$th millemeter-sized) vesicles. In some of the simple vesicles is found,—in place of a wall-inclined, secondary, spherical vesicle,—an oblong, wall-attached nucleus.

(*d.*) With these are associated many-layered, smooth, or slightly gibbous cysts, between the lamellæ of which oblong nuclei are often found inserted. The central vesicle occasionally holds a multitude of the most various primary and secondary forms,—elementary granules (nucleoli); spherical, oblong nuclei; simple and compound vesicles, and incrustations of vesicles. These laminated growths, for the most part, undergo an incrustation proceeding from the central layers.

(*e.*) We also find the cysts of the vascular plexus to contain, commonly in the shape of a *mucus-like* substance, deposited, as it were, from the fluid, and mostly infiltrated with fine sand granules, a transparent blastema pervaded with round and oblong nuclei. This gradually forms into areolar tissue, displaces the moisture, and ultimately fills up the entire space of the cyst, in the meshes of which the aforesaid forms all lie imbedded. Within the structureless blastema we perceive the oblong nuclei bent in accommodating curves around, and closely attached to, the vesicles (*alveolar textural arrangement*). Such cysts gradually shrivel around these their contents, and finally become extinct.

The aforesaid cysts are very delicate, commonly clear and transparent; some refract the light with a whitish tone, their contents appearing somewhat denser; in others the contents are of a reddish shade; in others, again, finely granular, as though coagulated; in others, lastly, the contents consist of a multitude of sharply-defined granules. To this class belong, more especially—

(*f.*) Globular bodies, in which the outline of a cyst-wall is wanting, and which represent lightly granulated spheres (*g.*) Lastly, the vascular plexus cysts often contain a whitish, chalky fluid. This consists almost entirely of fat-globules, and gradually thickens into a lardaceo-cretaceous pap, around which the cyst speedily shrivels, and perishes.

The vesicles and granulated spheres, the incrustations, the areolar tissue developments and the fat-globules, render the contents of the vascular plexus-cyst more or less opaque.

c. Finally, we have, in the well-founded expectation of throwing additional light upon cyst-development, examined their fluid contents. For this purpose we have found the contents of small (young) cysts, those, for example, which occur upon the broad ligaments of the womb, or within the cortical substance of the kidney, especially serviceable. They include a multiplicity of elementary forms, essentially identical with what are observed in the vascular plexus cyst. Nor is their occurrence limited to the cyst itself.

The fluid, semi-fluid contents of the sac, consist in an albumen-holding humour, which possibly presents various phases, the chief one being, however, that in which it constitutes colloid. In it are contained those elementary forms which are here fraught with peculiar interest. It is here adverted to irrespectively of what was before stated concerning its parenchymatous contents, as well as of whatsoever changes hemorrhage or exsudation may give rise to within the cyst.

Besides the débris of an epithelial layer generally composed of granulated, nucleolated nuclei, there are found :

(*a.*) Similar free nuclei, some holding two, three, or four nucleoli, some visibly exceeding the usual size of nuclei. Along with them here and there, a form of which it is problematic whether it be a nucleated cell, or a full-grown nucleus, with a nucleolus.

(*b.*) Granules, the larger of which being $\frac{1}{700}$th of a millemeter in diameter, are obviously vesicles.

(*c.*) To both are associated vesicles and cysts, which grow from the size of the aforesaid granules to $\frac{1}{33}$th millemeter, and, as will be seen, beyond this.

These cysts offer many points of interest :

a. They are simple, or else compound, incased within and within. As regards the latter, we meet in the first place with simple vesicles, within which a central granule or nucleus corpuscle has become developed. This progressively enlarges into a cyst, in which the same process may be repeated. In a cyst are often contained two, three, four, and more secondary granules, nuclei, vesicles, in which the process of ingeneration is still further repeated.

β. Their shape is commonly spherical, but often flattened by mutual compression. Some, owing perhaps to the inspissation of their contents, are wrinkled, bent inwards, more or less regularly indented. This applies to the simple cyst equally with the compound, affecting in the case of the latter all the involved cysts in various degrees, or it may be only one, and that the innermost.

Some frequently throw out various projections, prolonged into cylindrical processes, which in turn display ulterior promontories, or it may be inlets. This applies both to the simple cyst, and also to the compound, affecting all the layers in unison. Occasionally, the projections are obviously determined by endogenous development out of multiplex nuclei and vesicles.

7. Their contents present marked differences:

The contents of simple and compound cysts properly consist of a viscid fluid, in some cases translucent and colourless, in others, of a reddish tint. In compound cysts, these two conditions often alternate one with the other.

Some contain in and along with the said fluid, granules in various numbers, even to thorough repletion; some, together with these, clear, reddish-shaded vesicles, the size of a nucleus. Others contain granulated, spherical, oval nuclei.

The fluid contents of certain cysts appear denser, less transparent, opalescent; of others, still denser, dully transparent, presenting at the same time a marked cloudiness. In others, again, the density is still further marked, and the cloud more sharply defined.

This cloud results from a parting of the contents into spherical corpuscles, and flaky pellets of various size. In a given cyst it seems tolerably uniform, or else it consists in a radius-like fissuring from the periphery towards the centre of the cyst. In compound cysts, the separation differs in degree and relative amount in the individual cysts, being often more developed, either in the outer or in the inner cysts, and in one or other not present at all.

The fissuring presents much that merits attention. In simple vesicles or cysts, it extends from the periphery towards the centre, where the radii or the points of the wedges of substance, bordered by the fissures, converge. In compound cysts, a fissure present in the external vesicle, borders either upon the contour of a nucleolus, of a nucleus, or of the secondary cyst, in which, if there be a fissuring, it is independently constituted. Or again, the fissure-lines of the cysts correspond with one another—in other words, the fissure affecting the external cyst is prolonged through the second, third, fourth inner cysts, &c., with a radius common to all. This is rendered possible by the metamorphoses of the cyst-wall about to be explained.

With the condensation of its contents, the wall of the cyst gradually disappears, seemingly blending with the contents to a uniform mass. As in the case of the so-called cysts of the vascular plexus, the entire cyst is transformed into a dull, opalescent, resilient, simple or compound, colloid globule, which splits under the covering glass plate,—a spherical, oval, cylindrical, or nodulated colloid mass. To this category, doubtless belong the unexplained bodies seen by Kohlrausch in a renal cyst. (Vogel, Path. Anat.)

This relation of the cyst-wall further determines the contents, reduced, after the completed process of separation, to an aggregate of stellate flocculi, which break up into roundish, opalescent fragments of various size. There are always present globular débris from which numerous fragments of this kind have become separated.

This relation of the cyst-wall occasions the breaking up of the already fissured contents, either spontaneously, or from pressure, into stratiform, or wedge-like fragments, as is particularly frequent in incrusted specimens. The same cause produces the linear disruption, through pressure of the smooth compound colloid sphere, athwart many of its strata. These formations, together with the remaining amorphous colloid contents of the cyst, are sometimes tinged of a brownish or of a yellow hue, from imbibed pigment.

An ulterior change in these formations, which should here be noticed, is their incrustation with phosphate and carbonate of lime. It commonly affects the compound laminated cysts, but not unfrequently, the simple ones also; the comprehensive, equally with the small ones; the smooth, equally with the gibbous. It invariably emanates in the simple cysts from the centre, in the compound, from the central layer, whether this consist of a simple nucleus, or a group of nuclei in a vesicular nucleus-development. The secondary cysts lying side by side within a simple or a compound cyst, become incrusted independently of each other, and also of the parent cyst. The cysts affected with incrustation are those whose contents have undergone the condensation before referred to.

Lastly, many cysts contain granules and globules, which are shown to be of a fatty nature, the cyst resembling in some sort a colossal granule-cell.

(*d.*) There is in the primary and secondary formations within the cysts hitherto spoken of, frequently a colourless hyaline, or coloured colloid substance, in the shape of roundish, oval, facetted, poppy-seed, millet-seed, or lentil-sized pellets and flakes, and of larger misshapen masses. It is uniform in character with the opalescent, self-separating contents of the aforesaid microscopic cysts.

The presence of all the semi-transparent and opaque formations renders the cyst-contents whitish, or turbidly yellow. This portion of the cyst's contents is frequently separated in the form of a deposit.

With this I conclude the catalogue of facts, relating to the cyst. I shall now proceed to recapitulate these facts, and endeavour to elicit from them such deductions as they appear to warrant and uphold.

1. The cyst is, with respect to its organization and secretory function, an independent hollow growth, essentially based upon a substantive element.

2. At the characteristic turning point, between a primary (embryonic), and a secondary phase, overstepping the microscopic scale, the cyst consists of a structureless vesicle of from $\frac{1}{25}$th to $\frac{1}{10}$th of a millemeter in diameter, and an encircling fibrous layer, maintaining various grades of development. To these is added, as endogenous production, a nucleus or cell-formation, limited to an epithelial stamp. The cyst here completely resembles the glandular vesicle of the thyroid gland, and of the

supra-renal capsules. The encircling fibrous layer furnishes the *alveolus* for the reception of the structureless cyst.

3. The elementary germ of the structureless vesicle, resides in the nucleus,—nay, inasmuch as the nucleus is obviously generated out of an elementary granule, it resides in the elementary granule itself. The latter grows by intussusception into the nucleus, and the nucleus at once in the same wise into the structureless vesicle. The nucleus arising out of the elementary granule either retains the character of the latter, as a smooth, polished, sharply-pencilled vesicle, or acquires the well-known granulated character. It is obviously the former, in particular, that becomes developed into the structureless cyst, even the granulated nucleus, however, enters upon this development, its contents clearing up during the process, but resuming the granulated character afterwards.

This history of cyst-development is essentially corroborated by the expansion of the cell-nuclei, of so frequent, although by no means exclusive, occurrence in *cancer-cells*; an expansion first pointed out with precision by Virchow, but which, owing to the identity of development of the normal gland-vesicle, and of the cyst, cannot be regarded as heteroplastic. It consists in the development of the cell-nucleus into a comprehensive cyst, identical with that evolved out of the naked nucleus.

This corroboration is rendered complete by the fact, that, in the inflated cell-nucleus, an elementary granule present as a nucleolus, expands into a nucleus, generates within itself another nucleolus, and forthwith becomes a second cyst. This in my opinion affords important evidence of the vesicular nature of the nucleus, and of its evolution out of the elementary granule, through simple intussusception-growth which Reinhardt has shown to be a property of the chyle-, the lymph-, and the pus-corpuscles. (*See Virchow's Archiv.*, vol. 1, fas. 3.)

4. To the cyst in its primitive condition, as a structureless cyst, there accedes from without, and blends with it, a more or less marked fibrous texture. The cyst, in this secondary condition, consists of a wall of a definite texture, with an internal lining of epithelium, and is at once endowed with an enormous capacity of growth.

The structureless cyst is developed in a consolidated, structureless blastema, commonly studded with spherical and oblong nuclei, or else in a nidus of caudate and other cells. In the former case the cyst is speedily surrounded with a fibrous formation following the course of the encircling oblong nuclei, the cells contiguous to the cyst assuming an elongated, riband-like, caudate shape, and arraying themselves in parallel order around it. A remarkable tendency to enter upon this relation to the cyst is shown by the caudate cells, which constitute so many cancerous tumours. They first associate themselves one by one to very small, young, vesicles, and shortly overlay them in quite disproportionate numbers. Even oblong nuclei will fasten upon young vesicles of the kind.

5. This arrangement of the elements of a texture, brought about by the presence of young cysts, and consisting in an essential portion of those elements uniting to form capsules, and alveoli for the reception of the cysts, I have termed *alveolar textural arrangement*, or the *alveolar textural type*, and assigned to it a range extending far beyond the limits of a special heterologous formation. This establishes the distinction between the alveolar textural arrangement and many similar, but differently engendered, meshworks, cavernous structures and the like.

6. The cyst, in its primitive state as a structureless vesicle, and also in its development, fully corresponds with the simple gland vesicle—for example, *the thyroid gland, and its development*, as seen more especially in hypertrophy of the thyroid gland. Nay, the same anomalies of development, consisting in arrest and involution, are common to both. On the other side, the insensible progress of the gland-vesicle, when imbedded in its fibrous alveolus, from its normal standard to the morbid condition of a cyst, constitutes a process completely one with the secondary stage of cyst development.

7. Cysts form singly, or else collectively, in greater, often in redundant number. New cysts often arise within the fibrous wall of a parent cyst. There occurs also an endogenous multiplication of the cysts, new ones being developed in the fluid or parenchymatous contents of a cyst. In the former case, they do not in their development overstep the primitive condition, for lack of the adventitious element requisite to consolidate and advance the structureless vesicle into the true cyst.

8. The cyst, as appears under the circumstances discussed in

§ 6, possesses, under the same form, a different import. This it reveals more especially in the character of the textural elements frequently engendered upon its internal wall through the medium of the excrescences. These repeat, now a normal, now a heterologous parenchyma—for example, that of carcinoma. We are here reminded of Hodgskin's idea, which has, from time to time, been much too inconsiderately and hastily condemned.

9. Cysts are for the most part abiding growths, which often attain to a monstrous circumference. There are, however, cysts which never, or very rarely, exceed a certain volume, about that of a grain of millet, or of a pea, at which point they burst, void their glassy, mucoid, colloid contents, perish, and are substituted by fresh ones. *Exploding cysts*, to which belong the cysts constituting the so-called vesicular polypi, the ovula Nabothi. The cyst in its primitive state as a structureless vesicle appears to burst, in like manner, and eject the brood elements it contained. At all events, open vesicles now and then occur which do not appear to have become so by external means.

10. On the inner surface of the cysts are found *simple, bulbous or dendritic excrescences*, which represent a bulb-shaped, pouch-like, or a variously projecting, hollow-growth, branching out into secondary and tertiary pouches, and consisting in a hyaline, structureless membrane, studded with spherical and oval nuclei.

These bodies shoot from the inner surface of the larger cysts, isolated or collected in groups, sometimes from the innermost, sometimes, naked, from a deeper layer, and through slight fissures, or more spacious gaps, formed by the cyst-wall giving away. Or, coming from the deeper layer, they raise the internal structureless or striated lamina of the cyst-wall into a vesicle, which they afterwards perforate, and with which they coalesce, becoming blended together with them into a meshwork. In the smaller cysts they raise up the epithelium, and retain it as an investment.

They contain an albuminous fluid, or one possessing the germs both of physiological texture, and of heterologous parenchyma. Through its accumulation they become changed into protuberant or pouch-like sacs, which last are often patent at their free extremities. There is often developed within them a fibrous texture which imparts to them the character of considerable, fleshy, shallow-lobed (condyloma-like) tumours, as in the cyst of cysto-sarcoma; or they shrivel into fibroid growths and perish. Above all they frequently engender, in the terminal bulbs of their branches, young cysts, thus mediating the endogenous production of secondary cysts.

They frequently enter upon the final transformation into a stroma of fibrous texture, which receives into a meshwork or into alveoli the elements of parenchymata of various kinds.

They occur not only in cysts, but also upon serous and synovial membranes, upon mucous membranes, growing, in all cases, into the respective cavities. Their development frequently out of deep fissures in the cyst-wall, renders it likely that they spring at some depth out of heterologous parenchymata, and ultimately penetrate into larger spaces formed by the yielding of textures.

They appear everywhere as *germ-nidi, and as carriers of certain textural elements.* In the cyst itself their tendency is to fill up space by determining the production of physiological and pathological parenchymata, but in particular the endogenous multiplication of the cyst itself. In the vascular plexus, and in the so-called Haversian glands, they occur as physiological growths. They have sometimes the characters of a benign, sometimes of a malignant new-growth.

The correlation between the chorion-villi and the cyst-formation occurring within them in the shape of *acephalo-cystis racemosa of Laennec,* and between the subject under discussion, is hardly to be doubted. I have, however, failed, of late, to obtain fresh materials for investigating this point.

The cyst, in its primitive state, is subject to various anomalies productive of arrest of its development,—of involution of the sac. These anomalies consist essentially in changes in the contents of the young cyst; it is therefore desirable, in the first place, to point out what is most remarkable in the contents of the cyst generally.

The first and most marked phenomenon in point, is the presence of delicate, diaphanous, for the most part simple, but also nucleated vesicles, the one kind including reddish glistening, the other kind, colourless limpid contents. In growths consisting of several cysts, encased one within the other (expanded nuclei), it is common for one or the other to include reddish contents, whilst the remainder exhibit a colourless, clear, or slightly opalescent fluid. Occasionally these different contents alternate several times. These vesicles vary from the size of a nucleolus, or a nucleus, to that of a vesicle $\frac{1}{35}$th of a millemeter in diameter, or more. The small ones occur in all physiological and pathological fluids containing plasma;—in the blood, in the humor Morgagni, in exsudates, in the juice of various heterologous growths, in the grayish blastema supplying the place of atrophied nerve-medulla in the brain and spinal tube. The larger ones occur more particularly in conjunction with cyst-growths, and as forming part of the contents of cysts.

The parietes of the vesicle, whatever may be the nature of the contents, are uniform with the sheath of the nucleus. They resist the influence of acetic acid, or else the latter occasions thickening of the membrane, and a sharper contour of the vesicle. An examination of the contents is essentially facilitated by the following occurrence: In the above summarily mentioned fluids, there probably is always, freely suspended, a reddish glistening fluid or viscid substance, both in little globules, and in larger masses, moulded into various forms, and, where the current is impeded, divided into smaller portions. This substance is rapidly dissolved by acetic acid. It forms the contents of nuclei, and also of cells,—very commonly of cells the recipients of the brood nuclei of the thyroid gland vesicle, the brood nuclei of young cysts,—sometimes the contents of pus-cells, and of young cells generally. Here, again, it is, together with the very delicate cell wall, soluble in acetic acid. In like form we often observe, especially in the contents of cysts, a colourless, viscid substance, equally soluble in acetic acid, and probably identical with the ordinary colourless cell-contents.

These two substances are, to all appearance, unimportant modifications of the same material. Colourless elementary granules and nuclei, after evaporation of their surrounding watery medium, when placed under the influence of liquor potassæ, swell, and assume a reddish tint; we might, therefore, fancy that a difference in density determined the modification, the reddish gleam being simply due to a diminution of density.

The colourless (denser) contents are coagulated by the addition of acetic acid, becoming still denser, bereft of transparency, opalescent.

Akin to this change is that of conversion into a colloid substance.

Occasionally a fatty transition is suffered.

Let us now proceed to pass in review those phases of the cyst, as a structureless vesicle, which we have set down as anomalies of its development.

(*a.*) In the first place, numerous cysts, like the brood nuclei and cells they frequently contain, break up, in their primitive state, as structureless vesicles. This is occasionally preceded by a dehiscence and emptying of the vesicle. In other cases, the contents of the cyst are first converted into fat, a transformation of the cyst to a growth resembling the granule-cell.

(*b.*) Cysts developed within a fluid, [the fluid contents of a cyst], for lack of attachments, and of certain textural elements accruing to the structureless membrane from without, do not overstep the primitive condition. They succumb to the processes of involution already described, or enter upon changes hereafter to be explained.

(*c.*) A remarkable arrest in the cyst's growth attaches to the *endogenous* development of *secondary* and *tertiary vesicles*, resulting in those laminated cyst-growths destined to undergo early incrustation. The lamination may be restricted to a system of many-sheathed or encased vesicles, developed out of either central or extra-centrical, parietal nuclei. Or else several distinct laminated systems occur within a common peripheral one, several nuclei, simultaneously or independently developed within a secondary or tertiary vesicle, having expanded into vesicles, and these, again, generated their own proper central or extra-centrical nuclei. This explains the frequent deviations from the spherical or oval forms. The size of these growths varies greatly. We may have nuclei in which a nucleus-corpuscle is inflated to a vesicle simply bordering upon the contour of the nucleus, or, again, of $\frac{1}{5}$th of a millemeter in diameter.

(*d.*) Another arrest of the development of a cyst, fraught with its eventual destruction, consists in the conversion of its contents into colloid. The cyst becomes reduced to a sheathless colloid mass, becomes fissured or furrowed, and so broken up into fragments. In laminated vesicles, this transformation may affect all the vesicles uniformly or unequally, often a single, and that generally the central one, which, in this case, generates no further nucleus, so that all ulterior lamination ceases.

The consolidation to colloid affects the contents, not alone of the cyst, but also of its basis, the nucleus, and the elementary granule or nucleolus. This we observe alike where cysts are involved in the said conversion, and also under circumstances where no cyst-development takes place beyond a slight extranormal inflation of the nucleus. Such transformed elementary granules and nuclei occur in medullary cancers.

(*e.*) Next akin to colloid conversion is the *incrustation of the cyst,* and of its basis (the nucleus and elementary granule) with phosphate and carbonate of lime. It affects both simple and laminated cysts. These incrusted growths vary from the bigness of a nucleolus and a nucleus, to $\frac{1}{5}$th, or even $\frac{1}{2}$ of a millemeter in diameter. They are, however, most commonly of about $\frac{1}{25}$th to $\frac{1}{20}$th of a millemeter. They are identical in form with the simple and laminated cysts, namely, spherical, oval, smooth or nodulated, lobulated, spindle-shaped, or cylindrical, occasionally hourglass-shaped, trefoil-shaped, facetted.

On compression, they frequently split up into regular flaky or wedge-like sphere sections, a circumstance connected, no doubt, with the capabilities of colloid substances to split asunder according to determinate radial systems. At other times they break up into smaller roundish stellæ, or into wedge-shaped fragments.

In laminated cysts the incrustation is wont to commence with the innermost layers, where the colloid consolidation of the cyst's contents begins. From hence it proceeds to the external layers. Many incrustations are seen in which the outer layer is as yet free, the incrustation, as seen from above, appearing to surround it as with a light fringe. In vesicles with several collateral filial vesicles, the latter become incrusted—beginning in like manner with the inner layer—whilst the parent cyst and its layers remain free for some time longer.

It occurs originally either in molecular or in crystalline form, the completed incrustations displaying, now a stellate, now a crystalline, stratiform aggregation.

To sum up, it would appear that it is not the *cell* out of which the cyst becomes developed, but the *nucleus*, and that this latter again is evolved, through endogenous growth, out of the nucleolus or elementary granule. Lastly, that the structureless cyst wall is not, as we once supposed, an independent textural development.

12. SARCOMA AND CARCINOMA.

Kindred new-growths, important from their frequency no less than from the question arising, in every concrete case, as to their innocency or malignancy. They constitute an extensive group, comprising most of the so-called heteroplases. They occur, now as independent tumours, which usurp the place of normal textures, now as throwing out a multiplicity of roots or branches through the textures, interlacing with their elements, destroying these, and assimilating them to their own proper substance.

Solidified, and more especially fluid blastemata enter very largely into their composition, both becoming developed after their own manner into the elements described under the heading, "metamorphosis of blastema." They become wrought into a stroma, various in extent and in design, which forms the receptacle for fluid blastema and its elements. Along with the fibrous stroma developed out of solidified blastemata, massive fibrous textures, evolved out of cells and nuclei, are derived also from fluid blastemata. Lastly, the structureless parietes of parent cells, here so numerous, enter into an immediate fibrillation.

The ulterior development of the parent-cell determines that very common texture, the alveolar, as also the resemblance with certain gland-textures; for example, the salivary glands, the thyroid gland, the cortical substance of the kidneys, &c., resemblances which formerly and even recently gave rise to such designations as "sarcoma pancreaticum," &c. When these heterologous formations affect osseous structures, they often determine the extensive new-growth of a texture simulating bone.

Their chemical composition involves, besides the gluten of the fibrous elements, several other gluten-like (pyin-like) substances, along with albumen, casein, and fats.

In size they vary from that of a just cognizable new-growth to that of a human head, and upwards.

In form they occur as spherical, roundish, knotty, lobulated, stellate, irregular, ramified tumours. The form of infiltration belongs more especially to cancers.

Of consistency we meet with every gradation, from the fluid and gelatinous, to the consolidated, fibro-cartilaginous.

Blood-vessels enter into the fabric of all these new growths, although to a very different amount; some being, relatively to their volume, sparingly vascularized, whilst others are distinguished by their redundant vascularity. In those of the latter kind, the collapse of the blood-vessels after death occasions a marked change in colour, consistence, and volume.

These tumours have a pathology of their own, being especially subject to hyperæmia, hemorrhage, and inflammation with its sequelæ, more especially ulceration and necrosis.

Many undergo a process of involution and extinction, either spontaneous, or evoked by preliminary disease, especially by inflammation.

They originate at all periods of extra-uterine life, and are occasionally found of considerable size in the new born infant.

Upon the whole, no texture or organ is exempt from them. There is, however, something peculiar and remarkable in the affinity evinced by certain types for particular organs.

We would distinguish these new-growths, as shadowed forth in the heading, into two series, the one comprising *innocent*, the other *malignant* formations, for the grounds of which distinction we must refer the reader to the section on "organized new-growths."

We have selected the term *sarcoma* to designate the benign growths, not because of any especial analogy with muscle-flesh, but in order to fix and define a name familiarised by long usage, and also by no little abuse. The malignant we shall leave in possession of their ancient characteristic appellation *cancer*,—*carcinoma*.

To catalogue the species of these new-growths in a strictly philosophical order, is not feasible at this day. We shall best respond to the requirements of practice by determining the species upon the grounds of predominant structural and chemical relations, a due regard being had to the general habitudes of a growth. The varieties, numerous in the instance of certain species, are for the most part concerned with elementary texture, by which the general habitudes are not essentially influenced.

A. SARCOMATA.

These, as already stated, represent benign new-growths. They are always purely local affections, and therefore exist almost always as a solitary growth. They are most commonly due to mechanical influences; accordingly their seat is generally in organs or parts near the surface, and obnoxious to such influences. They are curable by complete extirpation; that is, they do not recur at the same spot, and still less do they multiply in other localities.

They generally constitute circumscribed, spherical, stellate, and clavate, superficially lobulated tumours. Often enough they ramify throughout the texture, nestling and luxuriating through the elementary parts, so that these perish, degenerating, so to say, into the heterologous growth.

They often increase to a very considerable volume, and this within a brief period.

They affect the areolar tissue, the fibrous membranes,—especially the submucous,—the muscles, inter-muscular tissue, and submucous muscular textures (uterus), the bones (osteo-sarcoma), particularly the facial bones, glandular organs, the mammary, the parotid glands, even the testicle; and, in rare instances, the brain.

The osteo-sarcoma is often inclosed within a skeleton sheath formed by the distended bone, which eventually becomes perforated. In rare instances a new-growth of bone enters, in the shape of an inner skeleton or frame-work, into its composition.

Generally speaking, sarcomata are more frequent than carcinomata in the early periods of life—those of childhood and of boyhood.

They seldom lapse into a process of ichorous ulceration spontaneously, although frequently through inflammation brought on by the membranous expansions which covered them, namely, the general integument or the mucous membrane, inflaming and sloughing away, so as to leave them denuded. This ichorous ulceration may even lead to cachexia and exhaustion; the inflammation itself, however, never gives rise to a specific infection, and to a multiplied production of the heterologous growth.

Sarcomata lend themselves naturally to a division into three species.

1. *Gelatinous Sarcoma.* — A very frequent heterologous formation, of which there are several varieties. It consists, besides some albumen, almost wholly of a gluten-, chondrin-, or pyin-like substance.

The varieties are principally referable to consistence, which varies from that of gelatin to that of fibro-cartilage, conforming itself partly to the amount of water held by the glutinous basement, partly to that of the textural, and especially the fibrous elements developed within it.

(*a.*) The first variety is a very soft, jelly-like, nearly limpid, tremulous, yellowish-gray new-growth, sparingly vascularized. It is the *gelatinous tumour*—the collonema of Johannus Müller.

J. Müller describes the parenchyma of collonema as made up of spherules, some of which are much larger than blood-globules, interspersed with crystalline needles.

We have met with the gelatinous sarcoma in different organs; more commonly, however, in the brain, and in the mammary gland, bearing, if not all the attributes of Müller's collonema, at least so close a resemblance to its texture as to remove all doubt as to their identity. Our specimens presented, on the one side, a perfectly embryonic form of collonema, on the other side one more highly developed, and bordering upon the following varieties:

A roundish, goose-egg-sized, gelatinous tumour, from the mammary gland, consists of a very soft, amorphous blastema, interspersed with, for the most part, very minute elementary granules, and delicate little twig-like fibre rudiments. This blastema is permeated by whitish membranous septa, differing from the basement in nothing save in their greater consistency.

A very bulky, lobulated, gelatinous tumour from the brain, displays branched fibres, resembling the elastic, with very numerously imbedded nucleated cells, mostly larger than the pus-cell.

One extirpated along with a portion of the inferior maxilla, exhibited a stroma, consisting of elastic, branched fibres, shooting forth twig-like, out of a stem.

Lastly, a fourth, from a spermatic cord, shewed single spiral, elastic-like, but transparent fibres, in an otherwise amorphous, tenacious blastema.

(*b.*) The second variety comprises a series of kindred new-growths, marked by a progressively increasing density and resistance, and mostly by a very pronounced stellate and lobulated structure. A white, areolar tissue-like fibrillation, cognizable with the naked eye, and bearing in its interstices nuclei and cells, caudate nuclei and cells, and nucleus-fibres, enters largely into its composition.

There is here often both a microscopical and also a ruder alveolar texture and cyst formation, which lend to the heterologous growth the semblance of a glandular structure; the alveoli and cysts being the especial holders of the gelatinous moisture.

This variety of sarcoma is generally endowed with considerable vascularity. During life, the new-growth exhibits various shades of redness, and offers to the feel either a woolly resiliency or a greater degree of elastic firmness. In the after-death collapse, it is of a grayish-red or reddish-white, flabby, and in various degress resistent.

(*c.*) The third variety consists of a firmish, amorphous basement, broken up into solid fibres, after the manner of the intercellular substance of hyaline cartilage, and teeming with cells more or less resembling those of cartilage. This variety approximates in its elementary structure to the cartilaginous new-growths, to the *enchondroma*, with which the gelatinous sarcoma is manifestly and essentially cognate.

These varieties, more especially the last two, are often found combined in one and the same new-growth.

The gelatinous sarcoma, besides the rarer localities specified in the instance of collonema, namely, the brain and the mammary gland, affects the parotis, the subcutaneous areolar tissue, the intermuscular parts, and with great frequency the periosteum and the bones, more especially of the face. The second variety is particularly marked by the immense circumference to which the growth becomes developed, often within a brief space of time, through redundant lobulation and ramification.

2. *The albumino-fibrous tumour; fibrous sarcoma.* This tumour is of a fibrous texture, and is distinguished from other and especially the pure, gluten-yielding fibroids, by its albuminous contingent.

The genesis of the fibres and their relation to the fluid albuminous blastema vary.

(*a.*) The fibre-texture becomes developed out of the fluid, that is the albuminous, blastema, according to the laws of the cell-theory.

(*b.*) The fibrous texture originates out of consolidated blastema, forming in this case a stroma, in whose interstices the fluid blastema is contained and becomes developed.

The fibre varies in all the forms derived from solid and fluid blastema. In particular, all the forms described under "metamorphosis of blastema," recur here as the stroma. Again, we have recognized in one of the forms specified in the section on "gluten-yielding fibroid," the transition form from the latter to fibrous sarcoma.

Varieties of this sarcoma, dependent upon the form and arrangement of the fibres, might, accordingly, be numerously adduced. A frequent appearance in the albumino-fibrous sarcoma, is the formation of the cystic and alveolar texture.

Just as, in the gelatinous sarcoma it is the gluten-like blastema,—so it is a predominantly albuminous blastema with which the fibrous parenchyma becomes drenched, or, with which the gaps of the fibrous stroma, the cystic and alveolar spaces, become occupied.

The albumino-fibrous tumour, like the gelatinous sarcoma, occurs in areolar tissue, in the periosteum, especially the submucous periosteum; and in submucous and muscular strata of areolar tissue, as fibrous, pharyngeal, nasal, uterine polypi, &c. Moreover, it affects bones, especially those of the face, and, more rarely, glandular organs, the parotis, the mammary gland, the testicle. It is spherical, elliptic, single, or more often lobulated and ramified, more or less resilient, vascularized, turgescent new-growth, with a texture fibrous to the naked eye.

3. *The albumen-like fibrous tumour.* (Johannes Müller.) The albumen-like sarcoma is a gibbous, tenacious, albumen-like tumour, sparely and but partially vascularized where its texture is slightly reddened and less firm. It consists, generally, of a white or yellowish-white, solid, fragile mass. Here and there it exhibits clefts or fissures, which contain a synovia-like fluid. It consists, in part, of a uniform, almost structureless or indistinctly fibrous mass. According to Müller, it is made up of a basement of multifariously interwoven, microscopic fibres, amongst which are interspersed a vast multitude of globules. The tumour is said to have yielded no gluten by boiling; the scanty extract to have been thrown down by the reagents of casein, and the insoluble main mass to have represented an albuminous body.

We have only once encountered this new-growth, namely, in the bone, within a loopholed, bony sheath.

A very interesting variety of sarcoma is

THE CYSTO-SARCOMA.

The combination of the heterologous parenchyma with cyst-formation, the ground-work of which is here, as elsewhere, the parent cyst and the alveolus, together imparting to the new texture the glandular aspect.

Just as the basis of the cyst in sarcoma is identical with that of the pure cyst, so, in like manner, do the types of both simple and compound cyst-formation here recur. In point of fact, therefore, the forms designated by Müller, as varieties of cysto-sarcoma offer a mere repetition of those types.

These varieties are:

1. *Cysto-sarcoma simplex.* The cysts imbedded in the parenchyma of the sarcoma, are on their inner surface smooth, or simply speckled with little, isolated, injected elevations of parenchymatous texture.

2. *Cysto-sarcoma proliferum.*—In the same parenchyma are found numerous intra-cystic cysts, some flattened along, others attached by a pedicle to, the parietes of the parent-cyst. With

A. Cooper, we have seen some of these secondary cysts as hollow appendices, or even free within the parent-cyst. In like manner parenchymatous masses of a fibrous, or of an acino-glandular structure, grow as pedunculate offshoots into the cavity of the parent-cyst. This offers a transition link to the following variety.

3. *Cysto-sarcoma phyllodes.*—A large clavate tumour, consisting of a firm mass, and presenting a fibrous torn surface. Within is one large cavity, or there are several cavities, unprovided with any cognizable proper membrane, into which firm, sarcomatous, red, vascularized, foliaceous, or warty, tufted, broad-based, or pedunculate, bulb-shaped, sometimes cauliflower-like, or fringed and villous excrescences, germinate and grow. These consist of the same substance as in the former case, but are mostly less dense and more succulent, readily drawn out lengthwise into fibres, or spread out to a membrane, showing that they exist in a folded and rolled up condition. Along with them the cavity contains a viscid humour.

In this description of the cysts in sarcoma, we easily recognize the types of pure cyst-formation. Whatever interest, however, attaches to this repetition, its description is inconclusive as regards the real nature of the new-growths. The main feature is still the heterologous parenchyma, which becomes the cysto-sarcoma without doffing its primitive character. This parenchyma is that of the gelatinous and albuminous sarcoma. A peculiarity, in point of form, consists in the membranous basement of sarcoma phyllodes, in its above-described exquisite form. Cysto-sarcomata, and the species phyllodes most particularly, often attain to a very considerable magnitude. They are frequent in the genital organs, of females more especially, in the mammary gland, in the ovary, less often in the testicles. In a word, they occur in all those organs specified as liable to the development of sarcoma, in its several species.

APPENDIX.

[Since the publication of Rokitansky's treatise on cyst and on goître, he has devoted much attention to the subject of *cysto-sarcoma*, the *chronic mammary tumour* of the late Sir A. Cooper, the *imperfect hypertrophy of the mammary gland* of

Mr. John Birkett, *the mammary glandular tumour* of Mr. Paget. Rokitansky, in an essay, read before the Imperial Academy of Sciences, at Vienna, in Jan. 1853, on "the new-growths of the mammary gland texture, and its relation to cysto-sarcoma," gives the details of several minute examinations of these tumours, the results of which are thus summed up: "In the one case are found imbedded within a small transparent, succulent tumour, acinus-like formations, consisting of a structureless membrane, and replete with nuclei. In the second case are found, in the texture of nodules, of which the tumour is composed, numerous delicate fissures bordered by a fringe, in which the matrix substance is, with the naked eye, seen to shoot inwards, in the form of almond- or bulb-like projections. There are also larger cavities present, into which stellate masses project. A magnifying power shows that these projections themselves begin to become lobulated at their terminal points. In other cases an extreme, lobulated, textural mass is thrown forward into a fibrous cyst; some of its lobes being again enveloped by a cyst. Upon the surface of others are found furrows, whose margins spring up into conical and bulbous excrescences, and, together with these, larger, open, cyst-like chinks. In the texture of those lobules are, moreover, channels and acinus formations, as also upon the cut surface, chinklets cognizable with the naked eye, which, under a magnifying power of 90 diameters, appear as considerable cavities branching out in all directions, whilst between their emissaries the neighbouring texture shoots inwardly in the shape of conical and bulbous excrescences."

It results from this that—

1. The acinus-like cavity with its emissaries resides in a layer consisting partly of embryonic, partly of fibro-cellular tissue.
2. The cavity enlarging, coalesces with the matrix substance, which grows into its space in the shape of conical and bulbous excrescences, whilst these, at their free extremity, throw out lesser projections.
3. As these multiply, the number of the fissure-like emissaries increases.
4. They shoot out from all points around the dilating hollow growth, or from individual points, or from one point only, into

the excavation. Where they are wanting, the dilatation of the cavity is uniformly that of a cyst with smooth parietes.

5. This dilatation is often very considerable, and the excrescences may also attain to a vast size.

6. In these excrescences, a secondary formation of acinus-like growths takes place, in which the events which occurred in the primary one are severally repeated. This occasions certain excrescences to appear encysted whilst others remain bare.

7. At the surface of the excrescences are observed furrows or open cyst-like clefts, into which excrescences intrude in like manner.

Dissenting from the view to which Mr. Paget inclines, namely, that these tumours originate as cysts, subsequently lose their cyst form, and continue to grow as solid masses, differing, moreover, with Mr. J. Birkett, who attributes them to a blastema effused into the areolar tissue of the mammary gland, Rokitansky contends that an acinus-like hollow growth which determines this tumour, and repeats one element of the mammary gland, becomes developed within a matrix of new-formed connective tissue, which primarily constitutes the tumour. And this tumour is by no means encysted, unless an adventitious fascia-like sheath of areolar tissue be called a cyst. A true cyst is only subsequently developed within it through the dilatation of the said gland-structure, the connective tissue layer being expended upon the construction of the fibrous cyst-wall. This, growing into the space of the cyst, and carrying before it the primitive structureless cyst-membrane, works out an encysted textural mass, an encysted tumour, in which the gland elements are reproduced.

The dendritic excrescences, therefore, which vegetate in the cysts of cysto-carcoma, are not in Rokitansky's opinion, and, as he once held, outgrowths from the internal cyst membrane, but intrusions into the cyst of its own cradle mass, or matrix, still invested with the primitive cyst membrane and its epithelium.

Cysto-sarcoma simplex, in which the cradle mass does not intrude at all into the cavity of the cyst, is of the rarest occurrence.

C. proliferum is engendered by the development, within the terminal excrescence-bulbs, of the acinus-like cavities into filial cysts, and the ingrowing of the cradle mass is here repeated.

The cysto-sarcoma phyllodes of Johannes Müller, with its amply developed, warty, cauliflower- and foliated- or cock's-comb-like ingrowths, has nothing to mark it beyond the size and development of the excrescences. The cyst-membrane is here no longer demonstrable, having coalesced with the cradle mass of the cyst.

It has been stated that the dendritic intrusions into the cyst may occur at one point only of the cyst, at several points, or, lastly, at all points simultaneously. In the last case they converge, coalesce, and eventually fill the entire cyst, determining thus its aggregate, lobulated structure.

With two exceptions, the one mentioned by Johannes Müller, the other by Mr. Paget, of these tumours occurring in the breast of the male, Rokitansky knows them only as effecting the female breast, where they are generally seated at the inner and upper part of the mammary gland.

Their figure approximates to the spherical. Small tumours are commonly even and smooth, greater ones irregularly nodulated, knobbed, lobulated, at the same time tolerably resistent, elastic, often generally or partially presenting the feel of a cyst tense with fluid.

The skin often presents a livid aspect, and is traversed by dilated veins. It is sometimes found coherent with the tumour, but not degenerated. The mammary gland becomes displaced by large tumours, and wastes away.

These tumours have an enormous capacity of enlargement, growing, now slowly, now rapidly, often with lengthened periods of arrest. Occasionally they disappear spontaneously. In a few instances several small tumours are concurrently present.

They are usually painless; there are cases, however, in which the pain is excessive. These represent Cooper's *irritable tumour of the breast,* a fibrous tumour (a neuroma) for the most part associated with the present new-growth.

The individuals affected are often unwedded, or childless females. The married and child-bearing are however not exempt.

The tumours are innocent, and, although often recurrent at the same spot after extirpation (Mr. Birkett relates a case of their reproduction five times in succession), not so beyond the range of the mammary gland. In many respects they are analogous to the fibrous tumour of the uterus, and to enchondroma.

In rare instances they undergo ulceration, which involves the superimposed cutaneous textures.]

B. CANCER.—CARCINOMA.

Heterologous growths not distinguishable from sarcomata by definite generic marks, and, like these, to be dealt with only as species, but contrasting with sarcomata in the single feature, common to them all, of malignancy. Carcinomata originate and subsist not rarely as local evils. Far more commonly, however, they are associated with a dyscrasis, which, in point of fact, often precedes and engenders the cancer. Hence the multiple appearance of carcinoma as the sequel to a single one, as the sequel to the extirpation of a voluminous and hitherto solitary one. Hence, in other cases, the original appearance of cancer in several organs simultaneously, or in rapid succession.

Conformably herewith, carcinomata can rarely with adequate reason be attributed to external local causes, whilst it is very common for them to luxuriate in internal organs beyond the reach of palpable influence from without.

The crasis which gives rise to the production of cancer, consists mainly in a preponderance of albumen, a defibrination (hypinosis), for the particulars of which we must refer to the doctrine of crases. Concurrently with this we have, more especially in the medullary crasis, an excess of fat in the circulating fluid, which determines a complication of cancer to be discussed in a more appropriate place; and, again, that remarkable relation of exclusiveness towards ordinary, fibrinous tubercle.

This crasis is essentially the same for all cancers, only exquisitely developed in the medullary form. This may be inferred, at least, from the frequent concurrence of various cancer species, in primitive or consecutive combination, either in the same locality, or in different organs. It may also be inferred from the circumstance that, after extirpation, the one is replaced by the other under the same contingencies, and that, conforming with an augmentation of the crasis, the medullary cancer is generally the consecutive one, more especially where the substitution takes place rapidly.

The highest grades of cancer-crasis originate through infection, that is, through the reception into the lymphatics, or more especially into the blood-vessels, of cancer-cells, or of cancer-blastema, of a lax, soft, semi-fluid character. The blastema is carried thither by imbibition, partly in the mere act of nutrition, partly, with or without the cancer-cells, through the lymphatics or veins laid open by ulceration of the tumour, or lastly, by the cancer penetrating into the canals of blood-vessels. Infection thus brought about, occasions locally, or it may be remotely, both in large blood-vessels and in the capillaries, coagulations of blood. In the former case, these are cylindrical, branched, plug-like, or else clavate coagula, adhering to the internal blood-vessel membrane, or to the endocardium (vegetations). They reveal their cancerous nature by their external medullary characters, as well as by their vigorous growth. In the capillaries the coagulation assumes the form of the cancerous depôt—so called metastasis (capillary phlebitis).

Cancer-formation assumes both a *chronic* and an *acute* course, the former being the more ordinary mode of occurrence for primitive cancer; whilst secondary cancer production is brought about with more and more rapidity in proportion as the cancers multiply. Ulceration and extirpation of carcinoma are especially apt to determine its very acute secondary formation. Still there are instances of highly acute, primitive, general cancer production. Moreover, the individual species of cancer manifest marked differences in this respect, both the first development and the ulterior growth, for example, of fibrous cancer, being slow, whilst in the case of medullary cancer they are incomparably more rapid.

In primitive cancers, the blastema is, in the great majority, insensibly produced. In acute cancer-formation it is thrown out under the symptoms of hyperæmia, and occasionally of inflammation. In the latter case, it often covers serous membranes with a stratiform cancer exsudate, or infiltrates and hepatizes the lungs with cancerous tubercles. From what has been said, our opinion may be inferred respecting the seat of cancer, in opposition to that of Carswell and Cruveilhier, who refer its origin to the capillary system. But, although in the ordinary process of cancer-formation we look upon the blastema as an exsudate in its broadest sense, we by no means question the origin of cancer from coagulation within the blood-vessels after the type of depôt-formation in general (see metastasis). It is indeed to this mode of development that we would ascribe the rapid cancer-formation engendered, in brutes, by the injection of cancer-blastema.

We are further disposed—although from isolated facts only—to believe in cancer-formation, through a conversion of certain physiological elements into those of cancer. In the liver, namely, we occasionally light upon a process, limited to circumscribed patches, of pallescence and alteration of the parenchyma, with some augmentation of its volume. Upon further examination, the portion of liver so affected is found to consist indubitably of hepatic cells, more or less bereft of their biliary and colouring matter, and of an intermediate, whitish, albuminous blastema,—as though the hepatic cell had become transformed into the cell of medullary carcinoma.

Cancers present sometimes well-defined, easily removable, spherical, irregularly knobbed, lobulated, branched tumours, which may lose their circumscribed character, only during their progress, by insinuating themselves betwixt the elements of textures hitherto merely displaced. Or they may appear, from the first, as infiltrated heterologous masses, involving the textures without definite limits. When an established cancerous mass stretches forth from one organ to seize upon a second, the latter is forcibly drawn in the direction of the first. Membranous formations, in particular, become attached to it with umbilical flattening, waste away, and become perforated by the heterologous mass. This is especially the case with fibro-carcinoma.

The size of carcinomata greatly varies. As tumours, some, and in particular the gelatinous and medullary forms, attain to a very considerable magnitude. A special notice is due to the occurrence of cancer in the shape of little millet or hemp-seed sized tubercle-like granules, as detected upon serous membranes. They have the import of medullary or of gelatinous cancer,—sometimes, however, of the alveoli, or follicles of alveolar cancer. In membranous formations, the magnitude is often represented in the superficial extension of the mischief wrought by the infiltrated malignant matter,—as in cutaneous cancer, in cancerous degeneration of the dura mater, &c. The number of cancers present in an individual differs materially, varying from the solitary tumour to almost general cancer-production.

With reference to the occurrence of cancer in the different organs, it may be generally stated, that no organ or texture,—not even cartilage—is exempt from it, with the solitary exception of horny textures. Certain organs, however, are hardly ever primarily affected with cancer, being attacked only, either under the conditions of general cancer-production, or through contiguity with, and by propagation from, some other organ previously a prey to the affection. Thus, *primitive* cancer of the salivary glands, or of the small intestine, very seldom occurs,—of the lungs or of the spleen scarcely ever.

As regards the preference of cancer for different organs, the following average scale of frequency might be established. First, the uterus, the female breast, the stomach, the large intestine, and especially the rectum; next comes the cancer of lymphatic glands, especially as retro-peritoneal cancer-accumulation in front of the vertebral column; hepatic, peritoneal cancer; bone-cancer; cancer of the skin, and of the lips; of the brain; of the globe of the eye; of the testis; of the ovary; of the kidneys; of the tongue, and the œsophagus; of the salivary glands and parotis. Again, we meet occasionally with cancers in large serous sacs, as in the peritoneum, for instance, adherent only by very inconsiderable portions of areolar tissue, or vegetating at large, and sometimes attaining to an enormous volume. Bone-cancer has now and then a sheath-like skeleton: far more frequently, however, it scatters the bony substance piecemeal, advancing at the same time, with redundant bone formation, in the shape of a laminated, stellate, thorny stroma.

Both in the primitive, and still more in the secondary appearance of cancers, we may,—apart from all disease from contiguity—discern certain relations of sympathy; for example, between uterus- and ovarium-cancer, between testicle- and kidney-cancer, between stomach- and liver-cancer, between stomach- and intestine-cancer;—cancer of the spleen is probably always associated with liver-cancer.

Generally speaking, cancer is more common in the middle and advanced periods of life. This relates, however, only to the cancer of certain organs, more especially of the mammary glands, of the uterus, of the stomach and intestines. All other cancers, especially those of the lymphatic glands, of the brain, of the eyeball, of bone, &c., occur even in early youth. Of the individual forms, the medullary is that most common at the earlier periods of life. In rare instances it is met with even in the fœtus.

Cancers are themselves subject to not a few diseases, amongst others, to hyperæmia with intumescence, and to hemorrhage. The most important amongst them is, however, inflammation. Besides this, they are liable to metamorphoses, inductive of an involution of the cancer; and these are developed, apart from external causes, in due proportion to the vascularity of the organ, and to the looseness of its texture.

Inflammation may become kindled spontaneously in the interior, the depths, of the heterologous growth. Or it may be the obvious consequence of external influences,—of irritating medication; of exposure of the tumour, after perforation of the investing external skin or mucous membrane, to contact with the external air or with passing secretions and excretions.

Its distinctive signs are identical with those which reveal the inflammation of normal structures, and they are developed in the direct ratio of the vascularity and looseness of texture of the heterologous growth. The disease has a marked tendency to open up, and, by hemorrhage and its results, to destroy such texture. It takes either an acute or a chronic course.

Its products are sometimes organizable; more frequently, however, and more voluminously, they are coagulable, yellow, fibrinous, or yellowish-white albuminous, deliquescent, pus-like, purulent, and ichorous,—very often hemorrhagic—exsudates.

Its terminations, besides discussion of the inflammatory stasis, and resolution, are:

1. *Abiding of the products* in their primitive crude condition, or else *disruption*, wasting, textural conversion.

We have here, first, to advert to the textural conversion of consolidated fibrinous exsudates, to gluten-yielding fibroid textures which may spring up as a reinforcement of similar stromata already in existence. Secondly, we have to mention the development of embryonic cancer elements out of a fluid exsudate, as represented in inflammatory hypertrophy,—increase of volume of the new growth. Both may concur with deliquescent, pus-like, ichorous exsudates, and, emphatically, with genuine pus-exsudate.

2. *Suppuration, ichorous production,*—the most ordinary termination. It runs either an acute or a chronic course, with or without simultaneous granulations possessing the character of a rapidly developed new-growth—representing certain lax, bleeding, easily suppurating, sloughing, fungus-like vegetations upon the cancerous ulcer. This process takes place either in the depths of the growth, in a shut space, as so-termed *occult cancer;* or upon the free surface of the body or of a mucous cavity, as so-called *apert or open cancer.* This last is, for the most part, marked by a funnel- or crater-like, deeply-extending base, with an elevated, mammillated brink.

Ichorous destruction of a cancer is very commonly followed by fugitive reproduction of the fungus upon the base of the ulcer, by the accumulation of cancerous matter in its vicinity, by cancerous degeneration of the implicated lymphatic glands, and, lastly, by the translation of cancer to other organs.

Even independently of suppuration, and without its concurrence, the necrosis of cancerous growths, both small and great, is not unfrequent.

Ulcerating and necrosing cancer—cancer-ichor—besides its corroding property, is marked by a very disgusting, penetrating fetor. This is, no doubt, essentially due to the sulphur and phosphorus of the broken-up protein and fatty constituents of the tumour, especially when exposed to the air.

We have here still to advert to one other important phenomenon. In cancers of the most different structure—in all cancers—we meet, not rarely, with a yellow substance, sometimes scattered in points, sometimes permeating the texture as straightened or serpentine, ramified striæ, interlaced to form a mesh- or net-work or, on the other hand, imbedded in considerable masses. It is a yellow, brittle, consistent,—or a soft, friable, unctuous, glutinous substance, which, if closely examined, is found to consist either of an amorphous blastema, dotted here and there with minute molecules, and interspersed with misshapen nuclei and with more or fewer of the elementary cells of cancer,—or else chiefly of scattered or grouped elementary granules (or fat-molecules), of the elementary cells of cancer replete with the same elementary granules, and lastly of fat-drops.

This substance constitutes, in the aforesaid mesh- or network, the so-called *reticulum* of Johannes Müller, who, regarding it as essentially prolific of cancer-cells, founded upon its presence a new species, under the denomination of *cancer reticulatum*.

On this point we cannot quite agree with Johannes Müller, the said *reticulum* not being confined to a single species of cancer, but occasionally met with in every form of the disease.

We hold it to be, generally speaking, a solidified product of inflammation destined earlier or later, to break up, its protein-substances along with the contained cancer-cells undergoing fatty conversion. We look upon this process as both interesting and important, inasmuch as, from its original foyer in the said substance, it gradually evokes a similar process throughout the cancerous growth. This is particularly the case where the substance possesses the reticular form, so as to master the cancerous parenchyma at all points. It is certain, however, that the cancer-blastema itself undergoes the very same transformation, and that spontaneously.

Cancers for the most part prove fatal, sooner or later, by their exhausting effects. The anæmia, emaciation, and eventual exhaustion are the result of the usurious growth of a single tumour, or of the development of a multitude of smaller tumours, or of hemorrhage or ulceration. Moreover, cancer, like other heterologous growths, kills through mechanical hindrance to the function of vital organs which it may have made its abode—for example, the brain. Acute, violent cancer-production rapidly destroys life, through the prefatory and attendant hyperæmia of important organs. Suppurating cancers become deadly through infection of the blood, and pyæmia.

However seldom the extirpation of cancer proves successful, its spontaneous cure is a still greater rarity. So favorable a result can only be brought about either by the progressive destruction, necrosis, and partial rejection of the tumour, or else by its more rapid death and expulsion; a circumscribing suppuration isolating it from the healthy textures—(mammary, uterine cancer).

Other processes of cure present, however, greater interest, bearing the character of an *involution*, a *decadency* of the cancer. Such are:

1. *Saponification of cancer*, a metamorphosis usually evoked by the conversion before alluded to of the substance constituting the reticulum. It partly consists in the liberation of fats, or in the conversion of protein substances into fat, with consecutive emulsive and saponaceous blending. This process, the above designation of which is warranted by a series of minute examinations, attaches chiefly to the encephaloid, medullary carcinoma so remarkable for its proportion of fats and of mutable crude albumen, and occurs more especially in the liver and the womb.

2. *Decadency, wasting of the tumour*, with condensation, solidification of its blastema, liberation of salts of lime in the shape of free molecule, and cell-incrustation. It affects in particular the denser cancers provided with a solid blastema (intercellular substance),—the firmer medullary and the fibrous cancers. The ossification and cretefaction of inflammatory products in cancer often gives the first impulse to this. Here again ossification and cretefaction (of the reticulum) are combined with fatty conversion.

Although carcinomata are, generally speaking, pre-eminently malignant new-growths, still the degree of their malignancy is not the same in all, medullary carcinoma occupying the extreme point of malignancy, whilst colloid and the epithelial cancers are in this respect the mildest of all.

1. COLLOID, GELATINOUS CANCER.

Alveolar Cancer (*C. Aréolaire.*)

In the array of cancers we again encounter a gelatinous, colloid new-growth, namely, *gelatinous cancer*, better known under the epithet *alveolar*, derived from its very frequent *alveolar fabric*. This texture cannot, it is true, alone mark the character of a species. It occurs, however, in gelatinous cancer so commonly, and at the same time in so exquisite a form, that under alveolar cancer nothing else is understood than gelatinous cancer.

This species occurs under two forms:

1. It presents a yellowish-gray, yellowish-red, here and there colourless, firm, jelly-like, transparent, tremulous, sizy, and when voluminous, irregularly clavate, lobulated mass. This consists of embryonic elements—for the most part a cell analogous in appearance to the pus-cell—in an amorphous colloid blastema, and of a very scanty, very delicate, fibrous texture, mostly investing the interlobular clefts. Blood-vessels enter into its composition only in very subordinate number.

2. *So-called* alveolar cancer—cited by Otho, in 1816, as a peculiar kind of stomach-scirrhus—cancer gélatiniforme, aréolaire of Cruveilhier.

The growth consists mainly and characteristically of follicles (alveoli) of either very delicate, pellucid, or of more compact and massive, white, satin-like, shining fibre-texture, containing a colourless or a pale yellow, grayish, diaphanous gelatine. Sometimes the growth consists entirely of these follicles, with inconsiderable columns of fibres intervening between them. Then, again, in the deeper layers, towards the base of the new-growth, we shall find the follicles separated by a copious, firm, white, fibrous stroma of new parenchyma. Its quantity stands obviously in an inverse ratio to the quantity and the grade of development of the follicles. This is shown most especially from the examination of cancers of the stomach and intestines. The walls are here found considerably thickened, hard, clavate, the inner layer presenting a multitude of collateral and superimposed hempseed- or pea-sized follicles, the innermost of which open, collapse, and cast their gelatine upon the inner surface of the stomach or intestine. In proportion to their depth, the follicles are, with exceptional patches, smaller, whilst the densely fibred stroma before referred to gains the ascendancy. This character of alveolar cancer is frequent both in the stomach and intestines, and may be designated as a combination of the alveolar with the fibrous form.

A closer investigation of alveolar cancer presents a fibrous texture of the parietes of the alveoli, and of the inter-alveolar substance. Along with areolar tissue-like fibrils, black contoured granule and nucleus fibres, with similar fibre elements arising out of solidified blastema and uninfluenced by acetic acid, play here a great part. In the gelatine are found, besides elementary granules and nuclei, non-nucleated and nucleated cells, cells with one or with multiple cells, parent cells. Besides these, there are present spindle-shaped, caudate cells,—under certain conditions granule-cells, together with a large proportion of fat molecule. Johannes Müller obtained out of this gelatine, by boiling, no trace of gluten. An alcoholic extract, boiled with water, contained but an inconsiderable quantity of a substance somewhat akin to ptyalin.

According to Mulder, the main constituent of alveolar cancer does not occur at all in the healthy body. We regard it as the same gluten-like substance which furnishes colloid (see Colloid), and which, as we see, constitutes numerous new-growths, both benign and malignant.

Cruveilhier further distinguishes a "cancer aréolaire pultacée," the follicles of which, instead of transparent gelatine, contain opaque pultaceous matter, in which Boutin Limousineau has detected casein. We hold this cancer to represent a transition state of cancerous gelatine to fat, with its ulterior saponaceous and emulsive combinations, partly in the act of granule-cell formation. The same transformation is witnessed in like manner in the first form of gelatinous cancer, and often affecting considerable portions of it. It is in its nature analogous with the so-called reticulum of fibro-cancerous textures.

Alveolar cancer displays, in a consummate form, all the characters assigned, under the head of "cyst," to the alveolar texture. In its most pronounced, that is, its most fully developed state, it offers the following varieties:

(*a.*) The gelatine accumulates in the follicles in so excessive a degree, that the walls of the alveoli, owing to the distension, become thinner and thinner until reduced to a mere residue. The heterologous mass degenerates into an almost uniform tremulous jelly, traversed by delicate membranous septa,—the residua of the alveolar walls. It is, in point of fact, scarcely to be distinguished from the first form of gelatinous cancer.

(*b.*) The follicles dilate with increase of substance of their walls (hypertrophy) into cysts, attaining thus to an enormous magnitude. This excessive development affects the follicles more and more, in proportion as they are more peripheral. The new-growth presents an aggregate of collateral and superimposed cysts, one or more of which, at the periphery, are of immense circumference. This growth resembles the compound cystoid, inasmuch as a redundant alveolar type is common to both, a circumstance readily ascertained in the instance of alveolar gelatinous cancer from the more and more dense fibro-alveolar structure exhibited on its cut surface, in proportion as its base is neared.

The contents of the enormously developed follicles include all the variations observed in cystoids, just as their parietes are subject to the same class of diseases.

The parts most liable to alveolar cancer are the stomach and the large intestine, the serous membranes and the peritoneum in particular, the omentum (less often independently than in association with cancer of the stomach and colon), the ovary, the bones, in rare instances the kidney, the uterus, and the liver.

Wherever situate, gelatinous cancer generally enlarges, and that often in a short time, to enormous masses. Upon serous membranes, especially the peritoneum, it occurs in scattered gelatinous accumulations, in little millet- or pea-sized tubercula, or in larger masses,—occasionally as a continuous bulky growth, which vegetates from a few points of adhesion only, if not almost free within the peritoneum.

Upon the peritoneum the first form is predominant, but not to the exclusion of the alveolar, the scattered tubercula having the character of isolated alveoli. In the ovary the cystlike alveolar cancer is prevalent, very often as encysted dropsy of the areolar cancer-form. This form occurs also now and then in the bones.

Gelatinous cancer, and especially the alveolar, is sometimes associated with other cancers. This combination, however, with alveolar cancer, must be considered apart from the alveolar type. This type constitutes every cancer an alveolar, but not a combination with alveolar gelatinous cancer. Every alveolar cancer may be regarded as associated with fibrous cancer, by virtue of a notable fibrous inter-alveolar substance. A combination with medullary cancer is generally brought about by the medullary cancer supervening upon the alveolar, the peripherous follicles of which fill with, and are eventually overlapped by the looser encephaloid mass; or else, the medullary cancer grows into the cavities of the alveoli. More rarely, the gelatinous cancer supervenes upon the medullary, in the cystic and alveolar form.

Pure gelatinous cancer is the least malignant of any, and, unless it prove exhausting by dint of surpassing volume, a dependent cachexia is less pronounced than in other cancers. It is very rarely the seat of inflammation and ulceration. It is for the most part solitary, although somewhat prone to extend to contiguous organs, and to scatter itself over serous surfaces, in the manner already adverted to.

2. FIBRO-CARCINOMA.

Simple Carcinoma.

The *scirrhus* of older pathologists, the only new-growth designated by them as cancer; other equally and still more malignant formations being by them divided into sarcoma and fungus. It is upon the whole the most compact in texture, and therefore the hardest of cancer-growths. Hence, the expression scirrhous hardness, formerly employed to denote in an organized product a resistance analogous to that of fibrocartilage.

In a parenchyma like that of the mammary gland, scirrhus commonly appears as a clavate, gibbous, indistinctly lobulated, somewhat branched, not sharply defined, very hard, grayish, or bluish-gray new-growth, which has the faculty of dragging down surrounding textures upon itself, is of moderate size, of from a walnut to a duck's egg, is heavy in proportion to its density of texture, and creaks under the knife.

Several deviations, to be hereafter specified, here present themselves. We shall, however, limit ourselves in this place to the statement that the density of the fibrous-texture, sufficiently cognizable with the naked eye, as also the hardness, do not always attain the presumed high grade. Under certain conditions, scirrhus becomes tolerably lax and succulent.

On a more minute examination, the principal mass appears to consist of a *fibrous texture*, imbedded in which are *embryonic* *elements*, in the shape of nucleus and cell. The former gives it the impress of fibrous cancer, and determines its density and hardness. The greater the number of the embryonic elements, the more does its fibrous texture serve as a stroma for a constituent, whose preponderating mass alters and determines its characters.

The fibrous ground-work presents manifold differences with respect to the form and the arrangement of the fibres.

1. Very commonly it is a fibre resembling an areolar tissue-fibre or fibril, or that of the organic muscles.

2. Sometimes it is a consolidated, tolerably transparent blastema, in the act of splitting into fibres and fibrils, and presenting a fibrous torn surface. In both instances there is an accession of granule- and nucleus-fibres in various numbers.

3. In a case of stomach-cancer it was a dense felt of black, branched, anastomosing fibrils, similar to the fibrils of fibrin.

With respect to arrangement:

1. The fibres for the most part point in one direction parallel to each other.

2. They radiate from different centres.

3. Considerable columns of fibres traverse each other at various angles, so that upon a parallel-fibred section we find displayed, here and there, the stumps of transversely and obliquely divided fasciculi.

4. The alveolar fibre arrangement is very frequent.

The embryonic elements consist of nucleus and cell. The former are often very numerous, as spherical, shining nuclei, furnished with black contours. Not rarely, indeed, the transparent nature of crude, cancer blastema makes it appear as if the cancer consisted exclusively of these nuclei.

The cells present many points of difference.

They are round, or angular, ganglion-globule-like, or again wedge-shaped, caudate, &c.

There are often present parent-cells, which become developed into alveoli; and upon a cut surface we meet with these, visible to the naked eye, in the shape of prominent, transparent vesicles, imparting to fibro-carcinoma the aspect of a glandular structure. Blood-vessels are not wanting in scirrhus, although their abundance is not very great. Lobstein is wrong in asserting these growths to be non-vascular.

Although the mass of fibro-carcinoma is not altogether dissolved by boiling, it yields, nevertheless, a notable amount of gluten. Compared with medullary cancer, it contains a smaller proportion of fat, (according to Martigny, a soft fat; according te Breschet, cholesterine).

Acetic acid certainly does, (although denied by Müller,) render the cells more limpid, throwing nuclei and nucleus corpuscles with black contours and some little shrivelling, more into relief.

No other cancer possesses, in so high a degree as the fibrous, the tendency to condense and corrugate the textures, in which it has taken up its seat, or to drag down upon itself contiguous, especially if they be membranous, parts. The invariable consequence is the wasting of the cancerous organs, and the shortening, with consolidation, of implicated membranous formations. Fibro-carcinoma is slow of growth, and slower in proportion as the fibrous character predominates in its fabric. It will thus vegetate long, without producing any visible cachexia, provided it do not interfere with the function of any vital organ, and provided it remain solitary. A more rapid growth is always conditional upon an overpowering development of embryonic elements out of fluid blastema; which latter, in the inverse ratio of its plasticity, relaxes the texture of the scirrhus, drenches it, and causes it to swell. It is often of a medullary (encephaloid) character, giving rise to a combination of fibrous with medullary cancer, in the shape of a more or less intimate blending of the two. With this, there is always a simultaneous increase of vascularity in the cancer-parenchyma, hyperæmious tumefaction, and inflammation; frequently, also, the development of a reticulum.

The presence of this reticulum changes fibrous cancer to that form which Johannes Müller has designated carcinoma reticulare or reticulatum. That is to say, we conceive ourselves to be warranted by experiment in assuming the latter to be fibrous cancer, plus the reticulum,—fibrous cancer in the aforesaid progress of rapid and redundant growth, and incontinently passing into congestion and inflammation. Its form-elements are identical with those of pure, fibrous cancer; although the embryonic elements and blood-vessels predominate. The capacious cells and membrane-clad cavities met with occasionally in carcinoma reticulatum by Johannes Müller, are probably nothing more than the follicles of an alveolar texture that has invaded the fibro-carcinoma; such follicles being replete with the substance of the reticulum as a product of inflammation. This so modified fibro-carcinoma frequently occurs in the mammary gland, attaining, for the reasons stated, a greater volume than pure, fibrous cancer.

Fibrous cancer occurs (primitively and in a developed form), in the mammary gland; in the stomach, perhaps, still more frequently; in the colon; in the submucous areolar tissue;— more rarely in the vaginal portion of the uterus, upon serous membranes, and in the subserous areolar tissue. Again, as an expansive degeneration of the omentum and of the mesentery; in the salivary glands; in the fibrous tunic of the bronchia. In several of these, as well as in other structures,—for example, the ovaries, the brain,— there occur cancerous growths of embryonic composition, and in all likelihood of fibro-cancerous nature.

With respect to shape, fibrous cancer in and upon membranous structures deviates from the clavate form before described. In the stomach, for instance, it represents degenerations, spreading along the course of the submucous, areolar stratum, and only here and there swelling into knobbed projections; whilst in the intestine it assumes the annular shape. Upon serous membranes, the pleura for example, it sometimes occurs as a fibroid exsudate, that is, as a densely fibrous, whitish, chagreened mass of unequal thickness, branching, as if outpoured, or dropped here and there, over the surface.

In the bones it appears in the shape of roundish knobs, imbedded in the diploe of the cylindrical bones, over which the compact covering plate becomes wasted by compression, giving occasion to spontaneous bone fractures.

In the frequent cases of cancer of the stomach we have the best opportunities for studying the character of the cancerous degeneration of muscular tissues. It consists in a development of white inter-fascicular striæ, imparting to the fleshy tunic a white-celled aspect. The white striæ consist of accumulations of nuclei, cells, and lastly, fibres, which receive and so to speak encapsule the swollen, reddish, or yellowish red, exsanguine muscle-substance. The formation of these septa multiplies, and they increase in volume until the muscle has entirely given way and perished.

Fibro-carcinoma is, for the most part, the primitive cancer in the organism, and very rarely indeed the secondary. The cancer-growths consecutive to it have, in proportion as they multiply, more and more the character of the medullary form. Even the occasionally more rapid development of fibro-carcinoma takes place under the supervention of medullary carcinoma, and the affection of the implicated lymphatic glands occurring in the consecutive series is of the same medullary character. In like manner the extirpation of fibrous cancer is generally followed by medullary growth.

In conclusion, we would advert to certain malignant accumulations, proved by antecedent circumstances to be undoubtedly cancerous. These infest bone, the ovaries, again the mediastina, the retro-peritoneal space, lastly, the intermuscular areolar tissue; and they are distinguished for the great bulk to which they attain. As regards their elementary fabric, they are almost always embryonic structures, that is to say, they consist of nuclei and spindle-shaped or caudate cells, which last, by their arrangement, impart to the whole the semblance of fibrillation. The inter-cellular substance (blastema) is very scant; and the heterologous mass is consequently very dense and firm. They are to be regarded on the one hand as embryonic fibre-cancers; on the other, as kindred with the firmer varieties of medullary cancer.

3. MEDULLARY CARCINOMA.

In every way the most malignant heterologous growth, described by Burns as *spongioid inflammation;* by Hey, and afterwards by Wardrop, as *fungus hæmatodes;* by Abernethy as *medullary sarcoma;* by Monro as *fish-testicle-like* (soft roe-like) *tumour;* by Laennec as *encéphaloide;* by Maunoir as *fongue médullaire.* All these appellations serve well to designate the external characters of this new-growth; that of fungus hæmatodes being, however, applicable to a combination of this malignant growth with redundant vascularity. (See new-growth of blood-vessels.)

If, for the sake of unity and clearness, we select for our principal delineation, medullary carcinoma in its most marked form, and with all the attributes of the most malignant cancer, we must preface the description by admitting that in the instance of no other cancer are more variations from this cardinal character cognizable.

In this, its exquisite form, medullary carcinoma certainly does offer a striking resemblance with the brain-medulla of younger individuals, or with the testicle of fishes; namely, a soft, semi-fluid, when present in large quantity, fluctuating, white, or under certain conditions, reddish-white or gray, yellowish-white, red or russet, or even in various degrees blackened, heterologous mass.

As an independent tumour, its cut surface exhibits either a perfectly homogeneous or else a variously cancellated, lobulated, more or less distinctly fibrous structure. When pressed or scraped, the cut-surface also yields a perfectly homogeneous substance out of a parenchyma which mingles with water to a uniform mass. Or, again, the entire mass separates into a looser medullary constituent, and into another more consistent, which furnishes a sort of stroma for the former, and appears as a more or less fibrous or villo-membranous frame-work. The relative quantity of both varies considerably.

These relations are subject to great variations, determined, for the most part, by the degree of consistency of the heterologous growth as cognizable with the naked eye. There are some growths of this kind which recede so far from the medullary character, as hardly at all to tally with the description above given of medullary carcinoma. Still, the occasional blending or interlacing of such deviating structures with exquisite medullary carcinoma, in one and the same organ, the embryonic condition of their elements, their rapid growth, and their voluminous character, seem to justify their mention in this place.

Thus there are, on the one side, medullary carcinomata of almost cream-like fluidity, or which, infiltrated into the textures, into the medullary system of the bones, or into the sheaths of organs after the destruction of their parenchyma,—for example, the neurilemma of the pituitary gland, the capsule of the spleen, &c.—resemble a milky juice. No stroma enters into their composition. On the other side, there are congenerous growths—heterologous masses, very commonly regarded as medullary carcinoma in a crude state, that is, in a primitive stage of the true medullary encephaloid—which, in point of consistency, do not yield to the fibroids, to fibro-cartilage. Amongst these denser masses there is one particularly remarkable—namely, an often very voluminous, in appearance, and also in reality, unevenly-lobulated, homogeneous, whitish, or yellowish-white, heterologous mass, which offers a striking analogy with the virgin mammary gland, especially in point of firmness and of elasticity. It is probably to this that Abernethy applied the term *mammary sarcoma.* Others present the aspect of a glandular structure; for example, of the texture of the salivary glands, or of the cortical substance of the kidney.

Lastly, the vast difference in blood-vessel-formation, referable to the structure of medullary carcinoma, is perceptible even to the naked eye. In no other parenchyma does it appear so frequently in redundance as in medullary carcinoma. Conformably herewith none is so susceptible of hyperæmia, of tumefaction, and of rapid growth; in none do hemorrhage (apoplexy) and inflammation so readily occur—processes, upon which the anomalous coloration of genuine white medullary carcinoma obviously depends.

The differences, however, discoverable with the naked eye in carcinoma, are slight compared with those revealed in the elementary texture of medullary carcinoma, with the aid of a magnifying power.

They are divisible into those recognized by the naked eye as components of medullary matter, and into those which, at the same time, present an intercellular substance,—a stroma.

With reference to the former, there are medullary carcinomata.

(*a.*) Consisting of granulated cells with a more or less distinct nucleus, and resembling pus-globules.

(*b.*) Consisting of smaller and greater, granulated, round, or angular, protuberant cells, more or less resembling the cells of tesselated epithelium, the hepatic cells, the glanglion globules, and provided with one or several nuclei.

(*c.*) Consisting of spindle-shaped and caudate, nucleated cells, fibre-cells, amongst which are many others, both spherical and oval.

(*d.*) Consisting of elliptical corpuscles, of $\frac{1}{100}$ to $\frac{1}{50}$ of a millemeter in circumference, and furnished with one or two nucleoli. They have the significance of a (heteroplastic) transcendent development of cell nuclei.

(*e.*) Consisting of spherical or oval corpuscles corresponding in size and tendency with the cell-nucleus.

(*f.*) Consisting of elementary granules down to the finest molecule-mass, with scanty nucleus formations in progress of development.

(*g.*) A further element concurrent with those specified at *b*, are pouch-like formations (see metamorphosis of blastema), and chiefly the parent-cell, which often constitutes a prominent element in medullary cancers. It forms here again the groundwork for the alveolar textural type of medullary cancer.

These elements occur predominantly, it may be, in the one or the other form but intermingled with others. Viewed with the naked eye, the elementary composition of a texture is, even to the well initiated, a matter rather of conjecture than of any certainty. The consistency and density of a texture may vary infinitely, being dependent upon the character of the intercellular substance. It is only where there is the appearance of fibrillation that we may perhaps infer a composition of spindle shaped or caudate cells.

Differences more important affect the character of the intercellular substance, and of a stroma in which the elements adverted to lie imbedded. This stroma is developed either out of those elements themselves, which, according to the laws of the cell theory, form into a fibrous skeleton work; or else it springs immediately out of a consolidated, amorphous, intercellular substance. Both together occasion, in medullary carcinoma, a special structure manifest to the naked eye, in the shape of a variously disposed fibrillation and lobulation, &c., the character of which so greatly modifies the consistency of the heterologous growth.

In this regard, we have the following forms, some more or less cognizable with the naked eye.

(*a.*) A medullary carcinoma, with an amorphous fluid, or semi-fluid, intercellular substance. The aforesaid elements vegetate in a thin or a thickish medullary juice. It is represented in the very lax, milky or cream-like *encephaloid* cancer.

(*b.*) A medullary carcinoma, with a solidified, amorphous, or else striated, indefinitely fibrous, intercellular substance, interspersed with roundish and fibro-elongated nuclei.

(*c.*) Medullary carcinoma, with a stroma consisting of fibre-cells (spindle-shaped, caudate) arising out of the development of the elements of the medullary substance itself, with consumption of the intercellular substance, and condensation of the heterologous growth.

(*d.*) Medullary cancer with a delicate hyaline, structureless, or else an opaque, striated, membranous stroma, studded with elementary granules and nucleus formations, or fibrillated like areolar tissue; which stroma, at the same time, forms the groundwork for the vascularisation of the alien growth. Its interspaces are filled with a loose, fluid, medullary matter, and it is easily thrown into relief if the tumour be scraped, pressed, or simply steeped in water. In villous cancer this stroma appears developed into a main constituent.

(*e.*) Medullary carcinoma with a more or less developed fibrous stroma, whose fibre-elements, upspringing from a solidified blastema, now resemble fibro-cellular tissue, now organic muscle fibre. It represents either a scaffold work or a stellate structure, the gaps being filled up with embryonic elements. Even with the naked eye it is discernible as denser striæ, disposed as aforesaid, and remarkable for their whiteness and their tendon-like lustre. This stroma has frequently the significance of fibrous cancer blended with medullary. It is, however, often enough an innocent fibroid growth, which may very possibly become the seat of so-called ossification (bony concretion). Hence the extraordinary phenomenon of medullary cancer becoming traversed by a concrete skeleton-work, in the midst even of soft parts.

This seems the proper place to take into consideration another combination with a benign new-growth in the shape of a stroma, namely, that with *normal bone-texture.*

A normal bone-texture occurs very frequently in medullary cancer affecting bones, as a thorny or stellate skeleton or stroma. This is, however, generally limited to the base of the alien growth. Greater interest attaches to a medullary carcinoma, possessing throughout a firm bony stroma, which, as a finely cancellated diploë, receives into its cancelli the soft parenchyma of the medullary cancer, to which it bears a relation similar to that of bone to its normal medulla. This growth certainly affects bones and their vicinity, although not exclusively. It is what Johannes Müller termed *malignant osteoid*. The bony texture entering so largely into its composition, is a very remarkable phenomenon, but its nature is simply that of a benign stroma for the reception of a cancerous, soft parenchyma.

An important part is assigned, in medullary carcinoma, to the parent-cell, and to the alveolar textural type resulting from it. We have often examined medullary carcinomata which mainly consisted of parent-cells. One consisted entirely of parent-cells, and being in the progress of fatty conversion, it presented a very peculiar aspect. Numerous liver cancers were found to consist of a fish-roe-like accumulation of yellow, poppy-grain-sized granules—parent-cells, replete with fat-containing filial cells—loosely connected together by a liquid, lardo-glutinous, yellowish-brown, intercellular substance.

Both forms of the alveolar texture occur in medullary cancer, the true alveolus, and also the aciniform, excavated body. Both, more especially, however, the latter, determine the likeness of many medullary cancers with gland-textures. Both may coexist independently of each other, or the second vegetate as an endogenous growth within the alveolus. Medullary carcinoma occurs no less frequently as cysto-carcinoma.

Upon the dura mater, heterologous formations are not unfrequent, which, closely resembling granular cortical substance of the kidney, consist of spherical or roundish rolls of caudate cells, imbedded in a layer composed of the same elements. They are gorged with a white medullary juice, are for the most part considerably vascular, and of a turgid, soft consistence.

Medullary cancer consists mainly of albumen, with fat, according to Wiggers a phosphorus-holding fat (brain-fat), according to Gugert cholesterine, and, as Eichholtz contends, with pyin.

Medullary carcinoma ordinarily assumes the form of roundish tumours; not rarely, however, both primarily and consecutively, that of infiltration into the parenchyma of every variety of organ. To the naked eye, the tumours often seem sharply sundered from the surrounding textures. Nevertheless, the impossibility of dissecting them out, without injury to those textures, and a narrower scrutiny, teach us that they penetrate into neighbouring textures, and, moreover, that they grow in suchwise as to infiltrate and destroy the textures in their immediate circumference. In other cases, however, they are capable of being shelled out of an organ, having a very delicate, areolar tissue-like, vascular sheath. Such growths are generally furnished with a membranous stroma, are more or less distinctly lobulated, grow independently, and simply justle the textures out of their place.

Medullary cancer in the one case grows to an enormous volume, in the other case is remarkable for its numerical dissemination. Its increase in volume, especially when rapid, takes place through the accession of embryonic elements. Hence the circumstance that old medullary carcinomata which suddenly undergo great augmentation of volume, have, at their base only, a solid and textural stroma, or it may be a bony skeleton. Medullary carcinoma is, both in its development and in its subsequent course, the most acute of all cancers. As a solitary growth in the organism, it arrives very rapidly at its full volume, and throws out a multitude of secondary tumours with the same celerity, not unfrequently under the accompaniment of very acute typhoid fever. The more hurried its development, the more does the embryonic form (elementary granule, nucleus, fluid intercellular substance) of its elementary composition, that of genuine encephaloid, predominate. Wherever cancer-production is acute, its form is the medullary.

Conformably with this, every other cancer, goaded into redundant growth, degenerates into the medullary, that is, enters into combination with the latter, the new accession being the medullary. The fungus upspringing from the ulcerating base of a cancer, is in its nature medullary. Every consecutive, every general, cancer-production is invariably medullary, nor is there any organ in which medullary carcinoma does not occur, either primitively or consecutively, as part and parcel of general cancer production.

In point of fact, medullary carcinoma occurs in organs in which no other cancer, least of all fibrous cancer, ever occurs; as in the liver, the kidneys, the lungs, the testicles, the lymphatic glands.

In the bones, medullary cancer is frequently distinguished by a lamina-stellate, thorny bone-skeleton, the form often obviously depending upon the nature and arrangement of the stroma. Sometimes it causes the bone to rise up into a bone-capsule; more frequently, however, it dissipates it into a voluminous honey-comb mass.

In medullary carcinoma the cancer-crasis has attained its *highest grade*. It experiences a further augmentation through infection,—through reception of this, the most readily absorbed cancer blastema, into the lymphatics and blood-vessels. The products of inflammation placed under its influence are eminently albuminous, white, opaque exsudates, and these become developed into medullary cancer, upon serous membranes, or as cancerous lung hepatization, and the like. The same thing happens with respect to coagula within the vascular system, both in the greater vessels and in the capillary system; by dint of an alienation of the fibrin, they bear evident marks of the cancerous character—cancerous phlebitis, capillary phlebitis (deposit).

We cannot subscribe to the assumption of regular stages of medullary cancer, of a stage of crudity, of softening, of ulceration, &c., these being conditions not correlated by any necessary causal links.

That which is regarded as crude medullary cancer, is the variety furnished with a consolidated intercellular substance. Softening obviously characterises the form of medullary cancer luxuriating as the true encephaloid, and it attaches equally to that which originates at once as such. Lastly, the ichorous and ulcerous destruction of the structure is a consequence of its inflammation, that is, of accidental disease of the tumour.

Medullary carcinoma frequently destroys life as a consequence of its surpassing growth, either as a solitary alien formation, or as one distributed over several organs,—through cachexia and exhaustion, through hindrance to the function of important organs; for example, of the digestion, of the larger veins,—the vena cava, by its closure; again through hemorrhage; finally, through inflammation and ulceration, often under the symptoms of cancerous infection of the blood.

The substance constituting the reticulum, occurs, especially in the softer forms of medullary carcinoma, in large accumulated masses. In the forms furnished with a fibrous or membranous stroma it follows for the most part the distribution of the latter, and therefore of its blood-vessels.

Contradictory as it may seem, after what has been stated, in no cancer is a spontaneous or natural process of cure brought about so frequently as in the medullary.

Such a process is the sudden and rapid destruction of the cancer by *ulceration* and *necrosis*, as observed not unfrequently in the dead subject, in medullary cancer of the womb. Such a process, again, is the metamorphosis described, under the general heading of "cancer," as *saponification* and *incrustation*. Moreover, it is known as a fact, that medullary carcinoma in the subcutaneous fat-layer will disappear through resorption, and return again.

With reference to the fungus hæmatodes of Wardrop, and the medullary carcinoma of Abernethy, we feel compelled to subscribe to Walter's verdict, namely, that they are identical. For we have always found the former to resolve itself, when closely examined, into medullary carcinoma with luxuriating vascularity. Assuming, therefore, the term "fungus hæmatodes" to designate a mere accidental condition of medullary carcinoma, there might be no impropriety in abandoning it, or in understanding by it only a highly vascularised medullary carcinoma.

On the other hand, it is requisite to bear in mind that which we have stated under the head of blood-vessel formation, namely, that assuredly there are alien growths, which, although primitively mere blood-vessel luxuriations, may subsequently combine with cancer, and this possibly without any concurrent anomaly of the general crasis, through mere impairment of the blood held within their own capillary system. We must here once more refer to the results of Van der Kolk's injections of the growths in question, which induced him to discriminate between fungus hæmatodes and medullary carcinoma.

That medullary carcinoma has some sort of affinity to the medulla of the nervous system, appears, not alone from its general aspect and chemical composition, but also from the fact that, in medullary cancer of the eyeball, the tumour springs from either the retina or the optic nerve, and that nerves speedily perish within the range of medullary tumours.

To medullary carcinoma we shall annex, *as varieties*, certain growths which bear an affinity to it.

(*a*.) CANCER MELANODES.

The entrance of pigment into the composition of any cancer converts it into cancer melanodes. Nowhere, however, does this substance occur in so marked a degree as in a cancer closely resembling the medullary. It may indeed be said, that *cancer melanodes* (*so-called malignant melanosis*) *is but a medullary carcinoma modified by pigment*, an idea promulgated by Meckel, von Walther, and others, in their day.

Cancer melanodes, as an independent tumour, presents most of the physical aspects of medullary carcinoma. Its cut surface appears to the naked eye either homogeneous, or fibrous, or lobulated, and of a more or less firm and brain-like consistence. A closer inspection of it reveals elementary granules, nuclei, cells of spherical or oval, caudate, elongated, angular shape, and along with these the most varied intercellular substances and stromata. Melanotic cancer imitates most commonly the encephaloid variety of medullary carcinoma, with round and caudate cells, and a membranous—a villo-membranous—stroma.

These alien-growths are chiefly marked by their black or brown-black, brown, bronze-green, or rust-brown coloration. The first glance at these often numerous tumours generally suffices to show that the colour is merely accessory. For, amongst thoroughly tinged, we meet also with perfectly colourless, white, heterologous growths; and again between the two extremes others pigmented in the most various forms, in dotted or stellate patches, or in ramifying anastomosing striæ. The white-growths are recognised at once as genuine, ordinary, encephaloid cancer.

A minute examination detects, according to circumstances, a greater or lesser proportion of pigment, and, even in the blackest, elements enough—cells and intercellular substance—free from pigment.

Pigment occurs free or inclosed in cells, in all the forms enumerated under that heading. Its basis is, as there taught, and especially as the examination of acutely produced or redundantly growing cancer melanodes incontestably proves, hæmatin in a free and dissolved state, or else blood-globules, with their pigment, in substance. In the latter case, the alien-growth resembles a hemorrhagic effusion, in which are found along with the blastema the elements of medullary cancer in various phases of coloration and of conversion into pigment.

Chemical analysis must needs detect the constituents of medullary carcinoma, and the pigment with its base. Barruel and Henry have discovered, in the melanosis in man, hæmatin, fibrin, three kinds of fat, a considerable amount of phosphate of lime, and iron.

Like medullary carcinoma, cancer melanodes is found to infiltrate the textures of parenchymata, as also of membranous parts, the dura mater for instance.

By reason of its pigment, melanotic cancer may be studied at its outset in very small point-like portions, which, under a magnifying power, appear minutely ramified.

Like genuine medullary cancer, the melanotic often attains to an extraordinary circumference. Its simultaneous occurrence in many, if not in most organs, is, however, still more usual. Its multiplication is often very rapidly brought about, with the concurrence, it may be, of acute typhoid fever. No organ is exempt from the disease. Even when attacking all, or several, organs simultaneously, it may grow inordinately in a single one or more than one, in which case the liver is almost always found to be the organ of predilection. We have seen it in the brain and about the nerves, at the eyeball, in the lungs, in the thyroid gland, in the liver, spleen, kidneys, bones, lymphatic glands, ovaries, in and beneath the intestinal mucous membrane, between the mesenteric layers, in the skin and subcutaneous areolar tissue, upon serous membranes, in the dura mater, upon and within the heart.

In the majority of cases, cancer melanodes is found to affect middle-aged or still older individuals. Both we ourselves and others have however observed it with little less of frequency even in youth.

The crasis upon which cancer melanodes is based, is without doubt essentially the medullary. The pigment has, however, still to be accounted for. A special dyscrasial character of the hæmatin and of the blood-globules might here suggest itself, a crasis analogous to the constitution of the portal blood with a continuous excess of aged and spent blood-globules which have reached their climax of coloration in a defibrinated plasma, the ready suscipient of hæmatin. Such a view would find support in the cachexia so often concurrent with melanosis, and so characteristic of a predominant venous constitution, with a livid, brownish coloration of the common integuments. And to this might be added the fact, that cancer melanodes is more than ordinarily rich in pigment when occurring in the liver and the choroid plexus, in which, for various ends, pigment is thrown out from the spent blood-globules even in the physiological state.

But, apart from numerous exceptions in this last respect, we must guard against overlooking very important local processes in cancer melanodes, where the base of the pigment is furnished, not by the general circulation, not by hæmatin, but by substantive blood-globules. Here the question is, first, whence is derived the blood as the basis of pigment? and, secondly, what causes the transmutation of the blood to pigment? The latter question is the more pertinent that in *medullary* carcinoma hemorrhage is common enough without any entailment of the pigment of cancer *melanodes*. In reply to the first query, we have to express a well-substantiated conviction that the blood furnishing the base of the pigment in cancer melanodes is not—at least not mainly—an extravasate out of a perfected system of blood-vessels; but blood newly formed in parent-cells, and transformed into pigment either within these cells or upon their breaking up.

This metamorphosis within parent-cells engaged in a process of radiation and ramification into a capillary system, explains the circumstance that the pigment, in its first manifestation in the parenchyma of a genuine white medullary carcinoma, appears in the form of finely-branched and stellate points and patches.

Cancer melanodes generally proves fatal in its excessive, multiple production, through the exhaustion and wasting corresponding to such redundant alien-growth. In rare instances cancer melanodes enters upon a process of ulceration, and kills through hemorrhage or simple exhaustion.

TYPHOUS SUBSTANCE.

The product of *typhous blood-stasis* deposited, in intestinal typhus in the follicular apparatus of the bowel, in broncho-typhus in the bronchial glands, and probably in plague-typhus in different superficial lymphatic glands, appears to us so analogous in many points with medullary carcinoma that we do not hesitate, in accordance with an opinion long entertained, to award it a place here.

Typhous substance appears, in extreme cases where it is rapidly produced under violent symptoms, as a grayish or whitish red, or a gray, or a white, lax,—in the mesenteric glands almost diffluent,—fluctuating, medullary substance, which, in its external features, bears the most striking similarity to encephaloid cancer.

This typhous substance, after abiding for a certain period in its primitive crude state, enters into a process of loosening up and sloughing, which becomes the medium of its removal from the normal textures. In some instances, and some epidemics, this breaking up manifests itself as a development of the typhous substance, both in the follicular apparatus and in the lymphatic glands, to a luxuriating, bleeding, partially necrosed, fungoid growth (Hensinger's muco-membranous fungus). The latter in particular, offers the greatest analogy with medullary fungus.

The elementary composition of the typhous substance is embryonic—elementary granules, nucleus-forms. Nucleated cells are commonly present in inconsiderable number. This relates, however, more especially to typhous substance in the bowel. That in the mesenteric glands frequently shows nucleated cells,—even parent-cells with several nuclei.

Even the albuminous constitution of the typhous substance, and the genuine typhous crasis itself, to which fibrinous exsudation is a stranger, involve an analogy with medullary carcinoma and its crasis. All fibrinous products occurring in the typhous substance itself, or along with it upon the same textures—the intestinal mucous membrane—or in any other organ, are not proper to the true typhous process, but to a secondary croupous crasis, into which the typhous crasis so often degenerates at various periods of its progress.

VILLOUS CANCER.

An alien-growth, whose cancerous nature is incontestably proved, both by its attendant cachexia, and by its frequent alliance with the cancers before discussed. Owing to the close affinity of its elementary structure with that of medullary carcinoma, we place it next in array with, or as a variety of, the latter; with which, moreover, it has in common the loose consistency, the abundant vascularity, and the proclivity to hemorrhage and to inflammation.

So far as we know, it occurs solely upon membranes, for the most part, the pituitous, and most particularly upon that of the urinary bladder, as so-called villous muco-membranous tumour. It also, although far less frequently, affects the common integuments and serous membranes.

At the outset, it appears as a delicate, cord-like excrescence of various length, which arises out of the aforesaid textures with a seeming longitudinal fibrillation, diverging at its free extremity into branches and twigs. Hereupon, if not before, it forms into delicately-membraned villi, and with this expansion of its texture, bulges at its free end into a club-like or cauliflower shape. This section of the excrescence invariably contains a whitish, or reddish white, encephaloid sap. At this point it is particularly vascular, and, in its recent state, of a purple tint.

A minute inspection shows the alien growth to consist of a fibro-membranous texture, densely involuted at the pedicle, and developed at the free extremity into a stroma for the reception of the imbedded encephaloid. This stroma is a delicate, structureless or striated, fine-fibred membrane, studded with elementary granules and nuclei, whilst the encephaloid sap consists of elementary granules, nuclei, and cells of every variety of form. Such excrescences not unfrequently vegetate in great numbers, either scattered or densely grouped, upon the mucous membrane of the bladder, imparting to it a long-drawn, villous aspect,—a condition ascribed by Andral to a preternatural development of the muco-membranous villi.

It is very common for them to vegetate particularly densely on a circumscribed patch, to become blended, at the pedicle and at the expanded points, into a diffuse, roundish head, furnished with a neck, which, if it contains much of the encephaloid juice, presents a uniform, pulpous consistency, and a superficial lobulation, whilst, in the opposite case, its periphery is villous.

The growth often bleeds spontaneously, and its excessive vulnerability occasions, upon very slight injury, exhausting hemorrhage.

From the above description, the medullary, cancerous nature of the alien growth is manifest, particularly its analogy with that encephaloid, medullary carcinoma, provided with a stroma. It is clearly nothing more than medullary carcinoma with predominant stroma-formation.

EPITHELIAL GROWTHS, EPITHELIAL CANCER.

These growths are without doubt often merely local, and curable by extirpation. In many cases, however, notwithstanding precisely the same morphological and chemical relations, they accord so entirely in all their manifestations with the cancers, that we classify them with these as a further variety of medullary carcinoma, to which in their lineaments, also, they approximate the most nearly.

Their occurrence we believe to be limited to the mucous membranes and the common integuments. We have seen them upon the mucous membrane of the larynx and trachea; of the stomach, the rectum, the urinary bladder; upon and in the common integument, and in the subcutaneous textures of the lips and face; in the scrotum, glans, and prepuce; in the external labia pudendi; upon the skin of the lower extremities. In a parenchyma we have met them but once, namely, in the liver, where they were encysted in a capsule of fibro-cellular tissue.

Upon mucous membranes these alien-growths usually appear as rather thickly pedunculated, roundish, cauliflower-like, or warty, leaf-like, stella-clavate, whitish, reddish-white, purple, vascularised, sometimes tolerably firm, often flabby, very vulnerable tumours, easily rent asunder by compression. Upon the common integument they sometimes form similar, now and then tolerably voluminous, tumours. More frequently, however, the alien-growth appears as a diffuse degeneration of the skin, which presents a warty, foliated surface, overgrown with luxuriating papillæ, or else, under different structural relations of the new-growth, a gland-like, sore, whitish-red, or red patch, which, under sloughing and offthrowing of the alien-growth, degenerates into one or several ridge-bound ulcers.

A more minute examination shows these out-growths to consist altogether of cells, which have hitherto seemed to us perfectly analogous, both in themselves and in their development, with the epidermidal or the greater epithelial cells of the tesselated structure. The mature cells are often of colossal size, flattened, mostly rhomboidal, furnished with one or two oval, reddish, or yellowish-red nuclei. The younger cells are smaller, roundish, spherical, limpid, or, around the nucleus, granulated in the figure of a sharply defined areola; whilst roundish, pale-red nuclei are present at their side. The older cells are of scale-like flatness,—their nuclei indistinct, or, it may be, completely obliterated.

In ulterior development the cell does not surpass—

(*a.*) A lengthening in one direction, with transformation to a rhomb or to a riband-like layer terminating at both ends in a short apex.

(*b.*) A parent-cell, within which occurs a second generation of cells, a development indicative of an alveolar disposition in the other surrounding elements.

These elements are held together by a very scanty, imperceptible, intercellular substance, and give way under moderate pressure, or without this, under the influence of acetic acid, or of other acids which serve to dissolve the intercellular substance.

The cells themselves manifest towards acetic acid relations varying with their age, the older ones not being changed, the younger ones becoming more transparent and gradually dissolved by it, whilst the nuclei are brought more distinctly into relief. When rubbed up with water they impart to it a whitish turbidness, and the young cells lend to their laxer bond-substance an encephaloid aspect.

The secondary arrangement of these elements is very remarkable. It consists:

(*a.*) In their arraying themselves in warty, or warty layer-like growths.

(*b.*) In their arraying themselves in cylindrical or facetted fibres or cylinders, which, gathered together into fasciculi, give the new-growth a fibred structure, a fibrous torn surface.

(*c.*) In alveolar order. Elongated cells of the secondary form above specified, course around circular gaps in which are impacted a brood of younger nucleated cells, either spherical, or, when very numerous, mutually compressed into polygonal shapes.

In the larynx, this formation constitutes the out-growths denominated by Albers warty, laryngeal tumours; many lax, succulent, seemingly fibrous, for the most part very sensitive, integumental, and subintegumental warts, a large proportion of cancers of the lip, scrotal or chimney-sweeper's cancer, a not uncommon condyloma-like degeneration of the glans penis, cancer of the external sexual organs in the female, and especially of the external labia. Many of these, more particularly cancers of the lips, have a seeming glandular texture determined by the alveolar type. From the common integument they assail subcutaneous textures without distinction,—even bone; from mucous membranes, the submucous textures; at the larynx, the arytenoid cartilages so commonly that one is induced to believe that the alien substance may in some cases originate with these.

Epidermidal cancer ulcerates, in the sequel of inflammation, in a form identical to all appearance with that of the most exquisite cancer. The base of the ulcer is invested with a yellowish-white, or a white, cream-like exsudate, consisting mostly of lustrous, reddish nuclei. Lastly, to this alien-growth is to be reckoned, without doubt, an ulcer developed out of a wart-like, transparent, hardish protuberance, in form thoroughly identical with ulcerating cancer, and not unfrequently seen to attack aged persons in the face. The base and edges of this ulcer consist of round, lustrous, reddish nuclei in an amorphous bond-mass, and the white, creamy exsudate investing the ulcer reveals the same composition. It represents embryonic stages of epithelial cancer. Certain epidermidal cancers of the lip are similarly constituted.

CARCINOMA FASCICULATUM,

(*Johannes Müller*).

Formerly termed, also by Johannes Müller, carcinoma hyalinum, because of its jelly-like transparency. An alien-growth, according to our observation, of very rare occurrence, which we have met with but twice; once in the mammary gland, and once again in most of the internal organs simultaneously, as almost general cancer.

The first case, which we had better means of examining, relates to a growth of considerable size, nearly that of an infant's head, of uneven, clavate surface, of a pale-yellow colour, of jelly-like transparency, and, withal, of notable compactness. It consisted of an aggregate of tubera, which resolved themselves into a certain number of cones, flat-sided from reciprocal compression, with their notched and ruffled bases directed outwardly, and their apices pointing to within, so that the apices of all the cones constituting a tuber converged to a common centre. The intersection between the individual tubers was occupied by a somewhat more substantial,—that between the cones by a more delicate,—membranous, whitish, areolar tissue-like bond-mass. Blood-vessels, so far as they could be traced in a not highly injected condition, ran in a direction parallel to the cones. A microscopic examination showed the parenchyma to consist of somewhat long drawn, delicate, hyaline fibres, between which, in an almost limpid juice, lay imbedded elementary granules, nuclei, and a few scattered, elongated cells.

Without conforming to Müller's description in what concerns the presence of embryonic elements, this growth accords with it, nevertheless, so fully in other respects, as to justify us in pronouncing it to be a true specimen of carcinoma fasciculatum or hyalinum.

In the other case, the secondary arrangement of the large conical fasciculi was less orderly, and, throughout, that before depicted, the consistence more lax, the transparency the same. This latter, according to Müller, is inconstant, and it was for this reason that he afterwards substituted for carcinoma hyalinum, the appellation of carcinoma fasciculatum.

The specimens examined by Johannes Müller were of a consistency analogous to that of encephaloid. He admits, however, that in this respect variations may occur, and that firmer specimens of carcinoma fasciculatum are probably to be met with.

CYSTO-CARCINOMA.

Cystocarcinoma specially affects certain organs, as the ovary, the mammary gland, the testicles, bones. It is mostly a growth of considerable magnitude, and commonly concurrent with cancer in other organs. [*See Cyst and Alveolus.*]

APPENDIX.

[A careful examination instituted by the author in sundry cancerous tumours, more especially of the medullary character, have led to interesting results illustrative of the development and the microscopic structure of these malignant growths. Without dragging the reader through the details of cases which seem only to represent so many stages of development,—so many links in the chain of evidence,—we shall endeavour to sum up the results in as few words as possible.

Under a magnifying power of 90 diameters, the substance of fungus hæmatodes exhibits a stroma consisting of two distinct webs, which appear to interlace each other in all directions. Of these, the one has the semblance of a transparent trelliswork, studded with caudate cells, elongate nuclei, and long-drawn fibres, all lying parallel to the longitudinal axis of the stroma. The gaps or meshes of this stroma are interlaced or enwreathed with what at first appears like a continuous garland of leaves, but on a closer inspection is seen to terminate in bulb-shaped extremities. Further examination shows this wreath-like tissue, which at first seemed opaque and granular, to be studded with crowds of minute nucleated cells, which, under a magnifying power of 400, are distinctly set forth as round or oval cells, many, although not all, containing one or several nuclei, others engaged in the act of elongation, others again in progress of dissilience.

In preparations representing a further stage of development, the wreath-like tissue presents certain patches much less opaque, its cells for the most part elongated, and many of its nuclei drawn out into disconnected fibres. There is good reason for regarding this portion as in a state of transition from the wreath-like tissue to the supporting stroma first described. In the next, and last, phase of development, is represented the same trelliswork, no longer thinly fibred and semitransparent, but rendered opaque by connected and dense longitudinal fibrillation. These fibred trellises are here distinctly seen to be enveloped in a hyaline structureless membrane, not closely fitting, but loose and projecting on all sides into the fenestrate gaps in conical and bulb-like excrescences. It is remarkable, that from the first period of their fibrillation, these trellis branches are observed to constitute hollow cylinders. This may be owing either to a single cell-layer being alone present within the excrescences, or else to the fact, that of a cell-mass with which the excrescence is replete only one layer becomes fibrillated and the rest absorbed.

Although the proof is difficult, there is good reason for believing that the hollow cylinders referred to are filled with the same cancerous substance that furnishes the outer material for the excrescences.

Certain external features, analogous with the above, induced Rokitansky to submit to a close investigation those adventitious membranes upon serous tunics, which present, with a honeycombed aspect, a free villous surface. The process of development resembles that of the cancerous growths, only that in these pseudo-membranous formations the wreath-like tissue more frequently occurs in layers parallel to the gaps or open spaces of the primitive fenestrate layer, or in superimposed order and sometimes in thick masses, tufted with many prominent, short-necked, terminal bulbs. It will be seen, from Rokitansky's great 'Essay on Diseases of the Arteries,' that the intra-arterial, super-imposed layers of coagula present very nearly the same structural development. (See 'Die Entwickelung der Krebsgerüste,' from the 'Sitzungsberichtie der math-naturw. Classe der Kais. Akademie der Wissenschaften,' März, 1852).

In a subsequent essay on villous cancer (April, 1852), and a third on colloid cancer (July, 1852), Rokitansky has made it apparent that, with certain modifications contingent upon the general conformation of the tumours and upon the nature of their contents, the same general relations of structure pertain to these cancers likewise.

In the *colloid cancer* there is a similar formation of a multilocular stroma, which however often assumes rather a *membranous* fabric. This honeycombed structure contains within its cancelli, the colloid or gelatinous mass, which is for the most part connected together, so as in a manner to interlace with the said stroma, and only here and there to occur in shut sacs or cystoids, formed through the blending of the membranous frame-work. Rokitansky has obtained evidence, that from the walls of these shut spaces, bulbous forms arise, and that the colloid globules are formed within these, as the product of a hyaline blastema with which they are more or less replete. He seems to infer that the fibro-membranous stroma is itself but a development out of primitive hollow bulbs.

There is in this theory respecting the aforesaid formation of the encysted masses of colloid, a general withdrawal by the author of one opinion expressed in the section on cysts, namely, that in these new-growths the cyst is invariably developed out of the structureless vesicle. The term *cystoid* would therefore be peculiarly applicable to the membranaceous cavities found in colloid cancer, as distinguishing them from genuine cysts.

Villous Cancer.—In all but its external form, this cancer approaches the nearest to medullary carcinoma. A very important part is here assigned to the dendritic excrescences, into which the primitive hollow bulbs, often springing from a densely reticulate germ, speedily resolve themselves; the first shoots pushing forth from their terminal bulbs secondary offshoots in the shape of slender villi, which themselves expand into bulbs, and throw out more of these embryonic excrescences from their terminations, so as to constitute by degrees a more or less extensive cauliflower- or coral-shaped tumour. In other cases, a single stem arises out of a nucleus as big, it may be, as a bean, and this stem branches out into dendritic vegetations of the character above described.

Most of these excrescences end in cæcal sacs, some of which may contain a structureless, or a concentrically stratified cyst.

These excrescences are often transparent, containing in their cavity only a clear fluid, whilst, externally, they grow up, as it were, into a more or less tenacious plastic mass, consisting of the same elements that compose the sap of medullary cancer. In other instances, they include a fibrous texture, within which reside elements similar to those that cling to them externally. A remarkable circumstance connected with these excrescences is the peculiar way in which they are vascularized. Both the stem of the tumour and all its individual excrescences are furnished with an ascending and descending blood-vessel, which pursues its course under the formation of frequent loops. These blood-vessels consist mostly of the primitive hyaline blood-vessel membrane, marked with oblong nuclei, sometimes also with a row of transverse oval nuclei. There may possibly be a further layer of connective tissue fibrils. A few of the excrescences have but a single ascending blood-vessel, terminating in a sort of bulb.

In rare instances, a nest of apparent excrescences displays open terminations fringed with villi, and filled with the semi-fluid materials of medullary cancer. Rokitansky is, however, of opinion, that these are not true excrescences, but rather lengthy developments of the fibro-cellular texture which constitutes the base of the tumour; and he believes these hollow cylinders, which seem rather to resemble the honeycomb of the wasp, to become filled, not by endogenous secretion, but by suction of the external medullary fluid.

Seat of Villous Cancer.—Its seat is more especially upon mucous membranes, and most of all that of the male urinary bladder, near the opening of either ureter; next to this, the mucous membrane of the stomach, and in particular the pyloric portion. It has been observed suspended by a pedicle from the internal membrane of the rectum, and even from that of the gall bladder.

Secondly, it is very apt to grow extensively from the internal wall of ovarian cysto-carcinoma, where it is recognised as villous cancer, from its copious accompaniment of medullary sap. In these cases, it is often concurrent with cancerous infiltration of the lymphatic glands, about the lumbar vertebræ, and with peritoneal cancer,—representing villous cancer upon a *serous membrane.*

It has been observed upon the dura mater, occasionally upon the general integument (Rokitansky refers to two such cases), and even in bone,—reckoning for villous cancer those cases in which a bony skeleton is found in the shape of the wasp's honeycomb structure before described.

Lastly, it occurs in parenchymata, in the uterus, for example; and, as cancer melanodes, in the liver and in the brain.

It occurs both as a single tumour, and also concurrently with cancer of various kinds in other organs,—occasionally germinating out of those broad based, fungus-like gelatinous cancer masses that occur upon the inner surface of the stomach.

"The vascularity of villous cancer determines a predominant feature in its course, whether upon membranous surfaces, in the interior of cysts, or in parenchymata, namely, the frequent hemorrhage which so greatly hastens the general wasting and the fatal issue. Frequent and excessive hemorrhage from the urethra in males, from the vagina in females, furnishes strong suspicion of villous cancer affecting respectively the bladder or the uterus, whilst a microscopic examination of the blood effused will often bring to light shreds or fragments of the cancerous mass."

The same vascularity often causes a fleshy coloration of the tumour.

It is evident from the foregoing, that villous cancer is, to all intents and purposes, a malignant *new-growth*: and not, as Andral and Louis have affirmed, an anomalous development of muco-membranous villi; nor, as others have more recently suggested, a tumour arising out of the hypertrophy of a pre-existent papilla.]

TUBERCLE. TUBERCULOSIS.

The collective term "tubercle" is made to embrace sundry formations, which have nothing in common beyond their outward form.

Still, after having well sifted this side of the question, we shall ourselves feel bound to comprise under "tubercle," formations in external appearance quite dissimilar to what is commonly called tubercle, nevertheless essentially identical with it; for instance, the primitively yellow, fibrino-croupous tubercle.

If we except the rare instances in which it represents an endogenous deposition within the circulating system, *tubercle* is in the broadest sense an *exsudate*—an exsudate of solidified protein substances (fibrin, albumen), which as blastema persists at the lowest grade of development; that is to say, in the primitive crude condition determined by its consolidation. It thus occupies the point of transition to the non-organized new-growths.

This last attribute is essential and indispensable, imparting to solid blastema the impress of tubercle. It is so important, that every blastema, however much its characters may assimilate to tubercle in other respects, loses the distinctive mark the moment it enters upon a transformation of texture.

This exsudate (in its broadest sense) is for the most part distinguished by the tubercle-form; that is, by its appearance as scattered or collected nodules, or where more copiously produced, by its deposition in granulations and stellate masses. It is hereby cognizable at the first glance. Still this is open to exceptions.

Gelatinous and fibrous cancer appear now and then in a tubercle-like form; that is, in the form of little discrete nodules or stellate bodies; and, upon serous membranes, the peritoneum, for example, there occur granular exsudates of fibroid and areolar tissues. These are distinguishable from tubercle by their texture.

But, again, even tubercle itself occurs in extensive, irregular masses. There are inflammatory products endowed with an indwelling tuberculous character, although manifesting a total absence of the external habitudes of tubercle.

Tubercle has therefore sometimes a local, but far more frequently a general import and significance. It is invariably so closely linked with dyscrasial processes, that, for a profitable consideration of tubercle, an incessant retrospect to the dryscrasial relations is imperatively demanded.

Nevertheless, the basis and starting point for an anatomical inquiry concerning tubercle itself, must in our opinion still be the aforesaid *fixed blastema abiding at its primitive stage of crudity*.

In this sense tubercle offers sundry distinctions, some obvious and essential, others less marked. They relate to its colour and lustre, its transparence, its consistence, its elementary fabric, chemical composition, &c. These are characters referable to more or less manifest special crasial relations—modifications of a fundamental tubercle-crasis. They determine several, and some of them essential, forms of tubercle, which we shall proceed at once to pourtray, selecting for our basis the purest possible forms.

(*a.*) *Simple fibrinous tubercle* appears as scattered or stellate conglomerations of granules of about the size of millet-seeds. It presents, moreover, as the product of inflammation upon serous membranes, smooth pseudo-membranous exsudates, as we often find exemplified upon the pleura of lungs involved in florid phthisis.

In the first-known form this tubercle represents the *gray semi-transparent granulations* of Laennec.

The investigations and theories hitherto instituted relate almost exclusively to this tubercle, from which all other tubercle-formations have been derived as from a stereotype basis.

The question of old—What is tubercle? must at this day be changed into—What is this particular tubercle?

In its early stage, at which acutely generated tubercle is often enough to be obtained in the human subject, it appears in the form of the aforesaid granulation,—to the naked eye a roundish, resistent, solid nodule, of about the size of a millet-seed. Not unfrequently, however, we encounter amongst them tubercles somewhat smaller, and representing a less firm, a softer, at the same time more transparent, almost vesicle-like granule.

Nevertheless, however much tubercle may at a first glance wear a vesicular appearance, it invariably originates as a solid corpuscle; and the results of a careful analysis of this substance, as well as its very nature and import, serve to corroborate this fact.

Minutely examined, it only seemingly represents a spherical body. Under a moderate magnifying power,—nay even on a narrow inspection with the naked eye, it is seen at its circumference to branch out more or less. With the textures it is only in so far connected as to lodge betwixt their elementary parts, to take up some of these into its substance, and—what is especially discoverable in tubercle upon serous membranes,—

to adhere to them by dint of an indwelling tenacious property. It represents a tolerably homogeneous—now toughish, gritty, fibro-granular, fragile, now softish, uniformly compressible—substance, in various shades and modifications of a pearly gray colour.

Under the microscope it reveals the following elementary composition:

It consists mainly of a more or less pellucid base (blastema), which affords a sort of binding medium for certain form elements. Its components therefore are—

1. The said basement-mass,—for the most part a fibro-glebous, gray, fixed blastema, rendered turgescent and transparent by acetic acid.

2. Certain embryonic form-elements, namely:

(*a.*) Elementary granules of various magnitude.

(*b.*) Nucleus formations, both black-contoured, lustrous, spherical, even oblong nuclei,—and more delicate, dull, granulated nuclei, under various phases.

(*c.*) Nucleated cells; commonly in such small numbers as to tempt one to doubt their occurrence altogether. Nuclei and cells are often to a great extent misshapen, disorderly, jagged, angular, bulging, dumb-bell shaped, rudimental, stunted.

Along with this, the tubercle is wont to include various elements appertaining to the textures in which it nestles. The tubercle purest in this respect is that upon serous membranes, which, therefore, like many other new-growths upon serous membranes, is the best adapted for examination. Nay, tubercle will even take up and incorporate compound textural constituents, and in particular blood-vessels. The question here suggested as to tubercle-containing vessels of its own will be discussed hereafter.

The metamorphosis which this tubercle undergoes, is limited to *decadence*. After abiding in the primitive, *crude* condition before described, it becomes transformed, with the loss of its moisture,—with condensation—to a hard nodule, and shrivels, into a tough, amorphous or indistinctly fibrous, horn-like mass, —in a word, *cornifies*. This determines a complete wasting and death of the tubercle, subversive of all further change. Occasionally this process is associated with bony deposition, the tubercle becoming a partly cornified, partly ossified nodule.

This tubercle does not undergo any other metamorphosis independently. Every other change suffered by it is based upon a combination of its blastema with another, and its *softening* in particular, upon a combination with the ensuing tubercle, namely, the fibrino-croupous. This *softening* process plays so momentous a part in the doctrine of tubercle, that we deem it right to declare emphatically our dissent from the opinion that gray tubercle, the gray tuberculous granulation of Laennec, softens.

Fibrino-Croupous Tubercle appears in the shape of roundish nodules, as also, and that very frequently, of irregular, gibbous, branched masses of considerable diameter, or, upon free surfaces as gibbo-stellate layers of various thickness. The nodules in size often equal the gray tubercle granulations, still oftener do they equal hemp-seed or peas. Usually, every variety of size coexists. The substance of this tubercle is, as we may here once for all remark,—opaque from the very first, now resplendent, in various degrees, yellow, of fibrous or of granular fracture, firmly elastic, or friable, of a lardaceous, curd-like aspect. We distinguish it from the gray tubercle by the designation of *yellow tubercle*. It most probably constitutes the pyin-holding tubercle.

The microscopic examination of this tubercle shows, as in the case of the foregoing one, a fixed base, and the aforesaid form-elements. The former is a fibro-glebous, or else an amorphous, opaque blastema. With respect to the latter much variety obtains. The number of cells, of nuclei, especially of the dull, granulated nuclei, of the elementary granules, and especially the quantity of the finest point-molecule predominate.

The metamorphosis proper to this tubercle is *softening*, and again *cretefaction*.

1. The first, namely *softening*, also termed suppuration, consists in this: after the tubercle has tarried for a certain time in the above-described condition of crudity, it loosens up,—for the most part with considerable increase of volume, readily breaks asunder through compression, moistens. Hereupon it changes into a yellowish, glutinous, fatty, tenacious substance,

like melted cheese, and eventually liquefies to a thin, whey-like fluid of acid reaction, wherein flocculent and fragmentary particles, the remnants of tubercle imperfectly broken up, float as tubercle-pus.

In the larger tubercle masses there is often observable, during the said process, a cleft formation on a large scale; or, where the tubercle is spread out in a layer, a fissuring of this latter.

With regard to the elementary character of the tubercle at this stage, we would observe:

The softening consists in a liquefaction and breaking up of the solidified base of the tubercle to a fluid loaded with point-molecule. This transformation results in a separation or isolation of the form-elements of the tubercle, which at the same time undergo within the fluid a more or less marked change. Thus, the cells become turgescent, corroded, dissolved; the nuclei shrivelled and misshapen, irregularly angular, pouched, &c. At length free fat becomes developed in the softened tubercle.

Hence the *liquefied tubercle* consists:

(*a*.) Of a fluid with point-molecule.

(*b*.) Of the isolated nuclei and cells changed in the manner just now specified.

(*c*.) Of free fat in the shape of elementary granules and larger scattered globules.

The softening determines the malignancy of tubercle, leading as we shall presently see to ulcerous destruction of the textures,—*tuberculous phthisis*.

2. The other metamorphosis of this tubercle is *cretefaction*. It never affects the tubercle blastema in its primitive condition, but only in its liquefying or liquefied state.

During the softening process, or after its completion, the tubercle takes up lime-salts and fats, in the shape of free, discrete or aggregated elementary molecule, or else in granule-cells in the form of big drops and of cholesterine crystals. In this act the softened tubercle is progressively thickened into a moist, unctuous chalk-pap, and eventually converted into a concrete mortar.

Let us now attempt to institute an inquiry respecting the *nature of tubercle*, in its two cardinal forms, as just delineated;

whereupon we will proceed to discuss its varieties, its metamorphoses, its local process of deposition, its seat, and, lastly, its relation to the blood-crasis.

In the first place, the ground-work of rapidly solidifying tubercle blastema is, without the least doubt, *fibrin*. Again, in the two cardinal forms of tubercle, it is easy to recognise the two principal forms of fibrin, the *simple* and the *croupous* (*see fibrin*). Why the former, which we have elsewhere denominated plastic, enters into no textural conversion, why the latter fails to undergo that prompt liquefaction proper to the croupous exsudates, are questions which we shall endeavour to reply to in a more appropriate place.

With reference to the varieties of these cardinal forms, we would observe—

(*a.*) Of *croupous tubercle* there occur *several varieties*, together reminding us of croupous fibrin and its resulting exsudates. They are determined by opacity, coloration, consistency, tendency to liquefaction, by the corrosive property of their ichor, the proportion of their form-elements, of their point-molecule, and by the character and import of their nucleus- and cell-formations.

(*b.*) *Like blastema in general, tubercle blastema is especially unwont to exsude pure.* The combination of the two cardinal tubercle-blastemata in different proportions, and their manifold grades of coordination and of blending; again, the union of varieties of croupous tubercle with each other, and with organizable blastema (fibrin), break up tubercle into countless varieties.

In like manner, the gray tubercle granulation presents many variations in respect to transparency, coloration, &c., the greenish shade, for example.

A peculiar variety is the *pigmental* tubercle, for the most part *hemorrhagic,* as to its origin.

As metamorphoses of tubercle, we have already been made familiar with its *decadence* or *obsolescence,* its *softening,* and its *cretefaction.* The first is proper to the simply fibrinous, the last two to the fibrino-croupous tubercle. These metamorphoses affect tubercle in common with consolidated blastemata of a certain constitution. whether they occur as exsudates (even as extravasate[1]) external to the vascular system, or as endogenous coagula within the blood-vessels. *Their cause is primitively inherent in the tubercle,* conformably with our view respecting the primordial properties of blastemata.

1. The *obsolescence* of tubercle, hitherto disregarded, is cosignificant with its cornification. It implies wasting, extinction of the tubercle.

2. *Softening* of tubercle, a metamorphosis which it enters upon without distinction of volume—resolves itself into that elementary phenomenon, the breaking down of solidified protein substances, and especially of solidified fibrin,—a phenomenon pertaining to this substance only in its determinate croupous constitution. It is proper to fibrino-croupous tubercle *alone,* and is determined by a conversion of the chemical components arising out of an interchange of the elements.

Genuine gray tubercle-granulation never softens. A combination of its blastema with that of fibrino-croupous tubercle alone capacitates it for softening. It was, indeed, formerly taught, that gray tubercle granulation lost its gloss, its transparency, became opaque, of a yellowish white or yellow, and ultimately softened and deliquesced. The error probably arose from a readiness, in the frequent cases where the two forms of tubercle are concurrent and even now and then mingle together into a kind of transition link from the one to the other, to take for granted that they represented in reality two different stages of development.

We have, however, a second error to rectify besides. Long ago the softening of tubercle was described as a development, —a progressive metamorphosis,—but in general and not very lucid terms. Present pathology, whilst adopting the older views concerning the softening of tubercle, is influenced by the microscopic discovery of an incomparably greater number of nuclei, and especially of cells, in *softened* than in gray tubercle. They are looked upon as new formations out of the liquefied tubercle blastema.

We cannot participate in this view. Those elements are not recently generated out of the liquefied blastema, but proper to the tubercle from the commencement, and isolated by the softening process. That they are more numerous in softened tubercle than in the gray, is explained by the fact, that only that tubercle softens which originally holds them in abundance, namely, the yellow (croupous) form.

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[1] The term "extravasate" is used by German pathologists in a restricted sense only, namely, to signify the effusion of substantive blood, with *blood globules,* into surrounding textures,—in other words, internal hemorrhage from ruptured or wounded blood-vessels.

In point of fact, no fluid is less adapted to furnish the blastema for new-growth than the so-called pus of tubercle. The softening of tubercle takes place sometimes early, sometimes late,—rapidly, or by slow degrees. All this depends upon certain peculiarities in the character of the (croupous) tubercle. In this process it is worthy of note, that in tubercle masses deposited all at once, the softening proceeds from the central part; whereas, in aggregate masses thrown out at different epochs, and perhaps embracing different forms of tubercle, the softening may commence at any part,—even at the periphery. This fact is fraught with interest, as corresponding with kindred processes in certain other morbid products,—for example, the central softening in globular endocardial vegetations,—in intra-arterial coagula-layers, &c. Moreover, it is important as offering—if at this time of day it be wanting—a conclusive argument against the assumption of the softening of tubercle being a process evoked from without through the agency of surrounding textures. The utter absence, in tubercle, of blood-vessels of its own, the compression and closure affecting such as penetrate the larger tubercle masses from without,—the fact that in textures surrounding tubercle engaged in incipient softening no trace of inflammation is generally discoverable,—that both the latter and suppuration supervene only upon completed softening of the tubercle,—lastly, the ocular proof that the softening commences at the point most remote from surrounding textures, are so many arguments against the assumption referred to, and especially against that of a mechanical melting down of the tubercle, through pus thrown out from the inflamed encircling textures.

The sum of these negations is, that the *softening is a spontaneous metamorphosis essentially proper to the nature of tubercle.*

The softening is that which constitutes (yellow) tubercle a malignant growth, inasmuch as it commonly leads to that ulcerous destruction of the textures which represents *tuberculous phthisis.*

The complete solution of a tubercle determines in the implicated parenchyma, a gap, generally corresponding to the tubercle in size, replete with so-called tubercle-pus. The parenchyma has suffered a loss of substance to the extent only of the texture particles which happen to have been involved in the tubercle, and have now perished in the tubercle-pus. This gap represents the *primitive tuberculous cavity* within a parenchyma. The contact of the tubercle-pus with the surrounding textures, occasions a corrosion of the latter. The moderate enlargement of the primitive cavity thus engendered, is substituted, upon membranous expansions, the mucous coats for instance, by a deepening destruction of the tissues; that is, of the inner stratum of the mucous membrane. This manifests itself as a millet- or hemp-seed-sized ulcer, which, to distinguish it from the different form arising from consecutive enlargement, has been designated as the *primitive tubercle-ulcer.*

The consumption of textures would here remain inconsiderable, but for the breaking down of fresh tubercle in the proximity of the original ulcer. Inflammation here plays an important part.

(*a*). This production of fresh tubercle in the vicinity of that softened, and of the resulting primitive cavity,—at the margin and base of the primitive tubercle-ulcer, upon superficial expansions,—determines the enlargement of the ulcer in all directions,—the textures becoming again and again corroded and necrosed by the fresh softening tubercle. And this takes place with a rapidity proportionate to that of the softening of the secondary tubercle—the product of an exalted cachexia. Another accidental mode of enlargement of the ulcer consists in two or more ulcers, already advanced in the way described beyond the primitive condition, merging in a single one. The result is an ulcer marked by its irregular, indented form,—upon mucous membranes, by serrate, jagged edges,—in muco-membranous canals by its affecting the girdle shape. The manner of its development, and its characteristic form, so different, especially on mucous membranes, from that of the primitive ulcer, fairly entitle it to the appellation of *secondary tubercle ulcer.*

The destruction of textures involved in this process, as corrosion and necrosis through contact with tubercle-pus, constitutes the *tuberculous phthises* of organs. These are either acute or chronic.

(*b.*) *Inflammation* enters, as we shall see by and by, into various relations to the phthisical process. What we have here, however, particularly to remark upon is in how far it contributes to the enlargement of the tubercle-ulcer and to the modification of its character. In the *first place,* it determines, for the most part, yellow tuberculous products, in the form of infiltration, which, conformably with the aggravated dyscrasis, rapidly break up, extensively corroding and destroying the textures involved. In this way inflammatory action occasions an ominous enlargement of the tuberculous ulcer, and the most widely spread tuberculous ulceration in an acute form.

In the *second place* it engenders organizable, solidifiable, fibrino- or albumino-gelatinous products, which pass into a fibroid callus. Thus arises the callous condensation of the textures encircling the cavity; in the muco-membranous tubercle-ulcer (for example, in the bowel) the hardish elevated brink and the funnel-shape of the *primitive*; lastly, the jelly-like infiltration and induration at the base and margin of the *secondary* ulcer.

These products exsude, according to circumstances, either pure, or almost pure, or combined together in various proportions. In worn-out individuals, the inflammation, if present at all, furnishes forth thin, albumino-serous products, and the tubercle-ulcer is of a lax and torpid character. In the proximity of cavities seated in the midst of tuberculous infiltration, there is of course no inflammation.

3. *Cretefaction,* as already stated, affects fibrino-croupous tubercle after it has entered into the softening process. It is co-significant with the cretefaction of fluid blastemata, and analogous with the cretefaction of broken-down fibrin in the vegetations and coagula within the vascular system, in croupous exsudates upon serous membranes, and in parenchymata; and again in the cretefaction of pus.

The basis of this (secondary) metamorphosis is as little to be sought extraneously to tubercle as the softening itself. Nevertheless, the surrounding textures may contribute, by their absorbing agency, to the inspissation of tubercle-blastema. What cornification is to the *gray*, cretefaction is to the *yellow* tubercle, namely a process of involution.

Cretefied tubercle resides, for the most part, within textures isolated by products of inflammation, entering into a fibroid transformation and then cornifying and shrivelling into a callous capsule. Both together draw down upon themselves the surrounding textures in scar-like corrugations.

Such are the metamorphoses of genuine tubercle of the one and the other form. There occur, however, *complicated metamorphoses* corresponding to various combinations of the different tubercle-blastemata. Thus:

(*a*.) The combination of gray with yellow tubercle is frequent. Where, in this combination, the latter passes into softening, the gray tubercle, like textures in contact with tubercle-pus, becomes destroyed. Where the softened yellow tubercle cretefies before this destruction of the gray is effected, the latter cornifies independently; and if it happen to be peripherous to the other it encircles the cretefied tubercle with a sheath of gray cornified tubercle, differing from that callous exsudate-capsule which results from inflammation of the surrounding textures.

(*b*.) Just as tubercle blastemata combine with one another, so, in like manner, does organizable blastema enter occasionally into combination with tubercle. Its existence is of course scarcely demonstrable in yellow tubercle, in the metamorphosis of which it becomes itself destroyed. If cretefaction set in early, it may become organized so as not to be easily distinguished from a subsequently effused blastema, the product of inflammation.

The combination of gray tubercle with organizable fibrin is more susceptible of proof. The instances are not rare in which, hard by pure gray tubercle, granulations are found in which one portion of their blastema is in progress of organization to a fibrous texture, whilst the other abides in its primitive condition, and eventually falls into decadence—cornifies.

There are, indeed, as we shall presently have to show, granular tubercles resulting from inflammation upon serous membranes,—that is, solidifying exsudates or granulations as big as poppy-seeds or millet-grains, which in their entirety change into fibroid textures—into areolar tissue. They occur, along with blastemata, consolidated into pseudo-membranous areolar tissue, or along with gray tubercle, or even with both gray and yellow, softening tubercle.

The organizing of these granulations consists in the development of a more or less determinate fibrous texture. They acquire the whiteness, the resiliency and elasticity, the fibrous-torn surface, the general characters of little fibrous tumours; or else they change into velvet- or felt-like fasciculi of connective tissue.

They are found upon the peritoneum, especially of the liver and spleen, as also occasionally upon other serous membranes. The Pacchionian granulations upon the arachnoid, the granulations upon the investment of the ventricles of the brain, are upon the whole of the same character.

It is intelligible, from hence, in how far, and in what sense, we are warranted in speaking of a textural conversion, an organization of tubercle, as a metamorphosis of this alien-growth. It is intelligible, namely, that growths, whatever resemblance they may bear to tubercle, lose their import as such, in other words, reveal their non-tuberculous character, with the slightest textural conversion.

Intimately connected with the above is the question as to *whether tubercle contains blood-vessels of its own?* The question may belong rather to a by-gone day. It is for the present day, however, to set this point at rest for all time!

Vascularity, in truth, belongs as little to the nature of tubercle as organization itself. Still it is undeniable that blood-vessels are sometimes met with in tubercles. Two cases are here possible. In the first case blood-vessels may appertain to textures which, whether normal or pathological,—membranaceous areolar tissue, for instance,—had become involved in the tubercle when first thrown out. One or more blood-vessels may traverse the tubercle, pervious to an injecting mass, —others impermeable.

In the other case the blood-vessels penetrating the tubercle are doubtless new-formed vessels, and have sprung from an organizable blastema, effused together with the tubercle and incorporated in it. This is attested more especially upon serous membranes, where, as a consequence of inflammation, tubercle becomes deposited along with a considerable portion of blastema, the rudiment of vascularized new textures.

To sum up: the *purer the tubercle, the more certain is its estrangement from all blood-vessel formation.* The less pure, that is, the more organizable blastema it has incorporated, or consociated with, the more susceptible is it of blood-vessel formation.

We have hitherto spoken of tubercle as being an exsudate,—a secretion from the vascular system; of which we hardly deem it requisite to furnish proofs. Here, however, a double question suggests itself, namely, first, concerning the *seat of tubercle;* and, secondly, concerning a very weighty point, namely, the *local process of tubercle production.*

The *seat of tubercle,* as exsudate, is at any point of any texture, extraneous to the blood-vessels. Wherever there is a capillary range, a deposition of tubercle is possible. The seat of tubercle is without doubt precisely, or at least in close proximity to, the spot of its exsudation, its blastema being in the highest degree coagulable. It is most probably for this reason that it does not affect textures nourished from a distance by a slow imbibition of their substance with plasma,—for example, cartilage. We can ourselves testify to the occurrence, both in larger blood-vessels and in the capillaries (as depôts or metastases), of coagula obviously of a tuberculous nature. These are, however, only exceptional cases, and the doctrine propounded in accordance with them is founded rather upon the results of so-called tubercle created by artificial injection. It is evident, however, that the tubercle-like depôts so formed were due either to infection of the blood, or to the obstruction of blood-vessels; and that no inference can be drawn from them as to the spontaneous formation of tubercle.

Assuming, therefore, tubercle to be an exsudate—an effusion out of the vascular system—the question as to the topical process would resolve itself into this: what are the processes in whose sequel tubercle is thrown off from the circulation? To this we can only reply, that tubercle, like other blastemata, exsudes, now almost insensibly in the act of nutrition; then, again, in the sequel to obvious (active) hyperæmia; and lastly, as a consequence of still more manifest inflammation.

1. It is a fact that the incipient production of tubercle takes place, within some organ,—most commonly the lung, and at one particular portion of it, the point de départ, so to term it, of tuberculosis,—in a manner almost imperceptible both to the patient and to the looker-on. The after-death examination reveals no inflammation, or such only as may with far greater probability be interpreted as consecutive. The tubercle is for the most part the gray, withering, and only now and then the yellow, softening, cretefying tubercle.

2. In other cases, on the contrary, a marked hyperæmia of the implicated organs manifests itself during life, and is discoverable after death, as the source of the tuberculous exsudation. The tubercle is deposited numerously, and also rapidly.

(*a*.) This tubercle has commonly the form of those scattered granulations, of about the size of millet-seeds, and seldom that of yellow hempseed- or pea-sized nodules.

(*b*.) Its blastema is commonly that of the gray tubercle, often combined with that of the yellow; rarely the yellow alone.

(*c*.) Not only is it thrown out rapidly and in great numbers, either all at once, or in successive outbreaks repeated at short intervals; but it scarcely ever restricts itself to a single organ, and whilst seemingly perhaps concentrating its main forces upon some one organ, it assails several others simultaneously, often leaving hardly any of the soft parenchymata unscathed. The tubercles are marked by their uniform size and character, and by the equable distribution with which they are scattered throughout the textures. After their repeated and copious exsudation, they gradually become less firm, softer, glutinous, until the fibrinous tubercle—the fibrin being expended—changes into the albuminous tubercle.

(*d*.) The effusion of the tubercle as a coagulable blastema is always associated with that of a non-coagulable or less coagulable, serous, sero-albuminous, jelly-like, adhesive product,—as a sort of vehicle for the first. The textures are manifestly congested; and, around the tubercles, more or less uniformly infiltrated with the product just referred to.

(*e.*) The more generally and more rapidly the tubercle-production extends through the organism, the greater the multitude of tubercles, the more marked those dyscrasial appearances wrought by defibrination of the blood,—by so much the more fully does the general disease partake of the acute character. As the expression of that defibrination, the blood appears thin and watery, the attenuated blood-serum, tinged with appropriated hæmatin, being thrown out upon and colouring the imbibed textures, which, if highly vascular, appear lax, flabby, and drenched.

(*f.*) As this tuberculosis for the most part proves quickly fatal, a metamorphosis of the tubercle is proportionately seldom observable.

(*g.*) Rare instances excepted, this tubercle is not the primitive one. Tubercle has commonly pre-existed, whether in an advanced state, or retrograding to decadence, in some organ or other; for instance, the lungs or the lymphatic glands.

3. In fine, *tubercle is frequently thrown out in the sequel of inflammation.* It is the product of such inflammation, and its sole distinguishing feature. These inflammations occur in every part, but more particularly upon mucous membranes, and in the larger serous sacs, where they may be most advantageously studied. Thus they are seen especially upon the peritoneum and pericardium, and again upon the mucous membrane of the uterus, of the tubæ, and of other ducts, as the vas deferens, the seminal vesicles, the ureters. They very frequently affect glandular hollow formations—the pulmonary cells, as pneumonia, the follicles of the intestinal mucous membrane,—almost equally often the parenchyma of the lymphatic glands, fungoid bones or sections of bones, &c.

The exsudate offers in regard to its so-called *tuberculization,* certain points of interest, as observed most particularly upon serous tunics.

(*a.*) In the first place, only a portion of the entire exsudate appears as tubercle, whilst the remainder becomes gradually reabsorbed and disappears. Or else this latter changes into a texture,—to areolar tissue, to a fibroid vascularized texture, often to a redundantly vascular, fibro-cellular new-growth, or to a spurious membrane of similar structure. The two allotments may be present in very different quantitative proportions, the one or the other predominating in various measure. The more texture-formation prevails, the more does the tuberculizing portion take the form of scattered miliary granulations, up to more voluminous nodules imbedded in the organized new-growth. The more scant the textural formation, the more prone is the tuberculizing portion to represent a confluent, uniform, granulating, stellate, clavate tubercle-mass or layer.

The tuberculizing new-growths, as vascularized pseudomembranes, often become themselves the seat of inflammation, for the most part productive of hemorrhagic exsudates of a tuberculous nature.

(*b.*) In the second case, the entire solid exsudate, remarkable for its bulk, is tubercle; or the organizable portion is imperceptibly small, and disappears. It forms in considerable, irregular, shapeless masses; or, on membranous expansions, smooth, or stellate, stella-clavate layers.

The tubercle thrown out as a consequence of inflammation, is the gray, or it may be the yellow, or again a combination of both. The fibrino-croupous yellow tubercle is especially often the product of inflammation, and especially marked by its abundance. It occurs everywhere, constituting, upon membranous formations, the aforesaid stellate layers; within mucomembranous canals and cavities of inconsiderable calibre, as, for example, the uterus, the tubæ, the seminal vesicles,—thoroughly closing plugs; in compact parenchymata,—lesser or greater, roundish or irregular knobs; in the lung cells and in follicles,—smaller coagula.

Tubercle produced by inflammation generally passes speedily into softening, and thus to a phthisis of the textures, marked by the acuteness of its course; and, as pneumonic tubercle infiltration, by the jagged, eroded look of the cavities; lastly, by the not unfrequent supervention of pulmonary gangrene.

Inflammatory tuberculosis, like the foregoing species, is rarely primary. It generally accedes to antecedent, insensibly generated tubercle, invading either the organ already diseased, or a structure intimately connected with it. Thus pneumonic tubercle and tuberculous pleurisy associate themselves to pulmonary phthisis; tuberculous peritonitis, to abdominal tuberculosis of the lymphatic glands. Or else tuberculoses become consecutively developed according to the same scheme, each fresh one bearing more and more decidedly the impress of its inflammatory origin.

The ordinary succession of the different fibrin-tubercles, and their different modes of exsudation, partly deducible from the preceding statements, are highly interesting. Generally speaking, the gray tubercle, insensibly effused into an organ, leads the way. The yellow tubercle is less frequently the primitive one. Nor is the hyperæmic or the inflammatory oftener the primary source of the effusion in either case. Upon the primitive, insensible deposition of gray tubercle follows, with increasing dyscrasis, the exsudation of combined gray and yellow tubercle, the yellow progressively assuming the ascendant, until it ultimately exsudes alone. Step by step the exsudatory process becomes more and more acute in character; hyperæmia, inflammation, and, at the same time, the quantity of tubercle thrown out, more and more pronounced.

Under certain conditions there exsudes, in the sequel of inflammation, more particularly in a new-growth naturally prone to tuberculization (pseudo-membranes upon serous tunics), a tubercle reddened and pigmented by adherent hæmatin and embodied blood-corpuscles. It might be suitably denominated the pigmented or hæmorrhagic tubercle.

The increment of tubercle, as a consolidated, non-vascular exsudate abiding in its rude, primitive condition, can only take place through adjacency and blending with a mass recently exsuded in its immediate vicinity. It is doubtless thus that many bulky, lobulated, stellate masses have become aggregated out of individual tubercles, dating from various periods.

But, whereupon does it depend that the product of the said processes, and, in particular, that the product of inflammation, in other cases reabsorbed or transformed into textures, is here precisely *tubercle*?

We do not consider the standing explanation of this phenomenon, namely, of the persistence of the exsudate in its primitive rude state, satisfactory. It is to this effect:

(*a.*) The lack of an adequate vitalizing influence in the surrounding textures, and in the entire organism, upon the exsudate (blastema). To this, it may be objected that the tubercle blastema *remains crude,* however scanty its proportion, and however unimpaired the energies of the surrounding textures and of the entire organism. On the other side, we find in the vicinity of diseased parts, and this in persons altogether debilitated and cachectic, bulky blastemata forming into textures both homœoplastic and heteroplastic; even into textures whose development is, generally speaking, very easily arrested; for example, bone callus in individuals affected with osteo-malacia, cicatrix in, and in the proximity of, ulcerating textures. We might with propriety ask, wherefore does not the portion of an exsudate farthest removed from the living textures and their influence,—wherefore, for example, in exsudates upon membranous expansions, does not an entire layer uniformly,—why, in the midst of exsudates, do only little scattered portions abide crude, that is, tuberculize, whilst the remaining major part of the exsudate becomes developed into textures?

(*b.*) The *lack of sufficient moisture,* of water, in the blastema, is alleged as the condition upon which tuberculization depends.

To this we reply that tubercle-blastema exsudes under all conditions, and, not at all rarely, with a considerable amount of water, of blood-serum. A primitive lack of moisture in the entire exsudate cannot therefore determine the tuberculization, the tuberculous nature, of its coagulable, solidifying portion. If perfected tubercle be poor in water, it is so obviously in consequence of the high degree of solidification of its blastema. This, therefore, namely, its high degree of coagulability, *might* be the cause of the tuberculous nature of the exsudate,—of its persistence at its primitive stage of crudity. This, again, might be the reason why the tuberculous exsudate takes the form of granulation. Inflammatory products, like blastemata generally, seldom exsude pure. Inflammatory products of a tuberculous nature are therefore, ordinarily alloyed with others of a different kind. Hence portions of the former emerging, by reason of their transcendent coagulability, from their combinations with the latter, appear to the eye in the shape of roundish coagula, in a word, of tubercle.

But, again, it will be necessary to ask, whereupon does this high grade of coagulability depend?

It can but be founded in an as yet unknown dyscrasial constitution of the fibrin, as tubercle-blastema. There are blastemata dry from primitive poverty in serum; and also others which, parting with their serum and passing into a high degree of condensation, nevertheless do not tuberculize, but become developed into textures, in the plenitude of their mass. It would appear evident, therefore, that the tuberculous nature of a blastema must be *indwelling,* be acquired either during the local process (inflammation) or in the general blood-disease which preceded and prepared its exsudation. Accordingly, tubercle would, as once before stated, have to be interpreted, now as a local, now as a general affection.

This *general character of tubercle* is the more marked in proportion as its mass as an exsudate is considerable; as its diffusion through the organism is extensive; as its characters are impressed upon any spontaneous coagula formed within the vascular system; and, lastly, as the organism in its totality reflects and manifests the tuberculous habit.

Let us now, as a sequel to the foregoing, discourse respecting *that anomaly of the crasis upon which tubercle is based.* With a view, however, to establish a suitable groundwork for the exposition of the tubercle crasis, we would first add a few supplementary remarks concerning tubercle itself.

The tubercle crasis is, without doubt, a fibrin-crasis—*fibrinosis.* It is not this in respect to quantity alone—hyperinosis—but also, and this is the more important side of the anomaly, in respect to quality. This is clear even from that varied constitution of tubercle upon which we have founded our classification of fibrin-tubercle. Besides this, the fibrin must have become impaired in a particular way, in order to qualify the tubercle, which, in the one case, as simply fibrinous, cornifies; in the other case, as fibrino-croupous, does not undergo the rapid process of dissolution (puriform liquefaction) proper to croupous fibrin.

This peculiar vitiation of the fibrin may itself become somewhat modified, or admit of some accessory impairment. And this may operate as the cause of many differences in tubercle, recognised to this day only by deviating physical properties; those, for instance, of coloration and lustre, of consistence, external form, mode of aggregation of the granulations, &c

Thus, the gray tubercle-granulation is distinguished at times by its dingy bluish coloration, by a grayish lustre, by its aggregation in sharply defined spheres thinly scattered through the pulmonary texture; the yellow tubercle by a lardaceous aspect. The croupous tubercle effused into the lung during the inflammation and ichorous offthrowing of cancers, is, owing no doubt to the fundamental cancerous vitiation of the fibrin, remarkable for its whitish coloration, its softer glue-like consistency, its liquefaction to a whitish, cream-like ichor.

A point of great moment, in relation to the crasis, is the recognition of an impress upon general nutrition indicative of a predisposition to tubercle, and consisting in certain developmental proportions of textures and organs; in a word, the "*tuberculous habit.*" Another point of equal interest is the relation of tubercle to other morbid processes, bound up with primitive or consecutive anomalies of the crasis.

There exists undeniably a habit, expressed in a delicate construction of the soft parts, in imperfect development of the muscular, with preponderance of the vascular, system, and especially in a so-called phthisical build of the thorax, commonly deemed ominous of pulmonary tubercle. It is essential, however, that this build should not, according to the vulgar notion, be imputed to smallness of the lungs within a seemingly insufficient thorax, but rather to very voluminous lungs within a thorax, the obvious narrowness of which, in its antero-posterior diameter, is amply compensated for by its length, with a relatively abdominal cavity, and small abdominal viscera.

Nevertheless, tubercle does not always, nor exclusively, thrive upon a substructure like this. The tuberculous crasis, like the local tuberculosis of an organ, may become acquired in an individual of quite a different habit, as a consequence of surpassing external and internal mischief.

With reference to the second point, namely, the relation of the tubercle to other morbid processes, no disease offers so much that is interesting, that is corroborative of views already set forth, that is practically serviceable and inductive of ulterior research, as tubercle. It is especially distinguished by its exclusive relation towards several morbid processes.

The sum of an immense range of experience in point is to the following effect:

1. *Cyst formation*, as a new-growth, is rarely found concurrent with tubercle, either in the same organ or in the same organism generally. In this sense the proliferous cyst-formations are distinguished. Where their seat is in the abdominal cavity, as for instance in the ovary, the immunity against tubercle is augmented by an accessory circumstance, in itself most important, namely, the coarctation of the thoracic space by pressure from beneath.

Experience seems to show that it is more common for cyst formations to succeed to the extinction of tuberculosis, than the converse.

A comparison of the occurrence of both in the various organs, establishes, as the extreme points of the scale, the well-known great frequency of tubercle against the extreme rarity of cyst formation in the lungs, and the reversed proportion of the two in the ovaries, and next to these in the salivary glands. This relation seems highly important in reference to the affinity which cyst formation bears to sarcoma and carcinoma.

2. A similar antagonism, as shown from still more numerous observations, prevails between tubercle and carcinoma. Whenever their general correlation is susceptible of proof, cancer has seemed to succeed to tuberculosis, tubercle rarely to become developed after the extinction of cancer and its crasis. Moreover, it must be repeated that to cancer, and in particular to inflamed and ulcerating cancer, there is sometimes superadded, more especially in the lungs, a tubercle, marked by a whitish coloration, a softish glue-like consistence, and a tendency to resolve itself into a whitish cream-like ichor. It has the import of tuberculo-croupous impairment of a carcinomato-dyscrasial fibrin.

A corresponding result of much interest is afforded by a comparison of the scale of frequency of cancer and tubercle, as well as of several special local relations of both.

They are diametrically opposed to one another, as thus:

FREQUENT.	RARE.
Lung tubercle.	Lung cancer.
Ovarium cancer.	Ovarium tubercle.

FREQUENT.	RARE.
Salivary gland cancer.	Salivary gland tubercle.
Stomach cancer.	Stomach tubercle.
Œsophagus cancer.	Œsophagus tubercle.
Rectum cancer.	Rectum tubercle.
Ileum tubercle.	Ileum cancer.
&c.	&c.

Again, the special localities present many differences of their own. Thus, in the uterus, the vaginal portion and cervix become affected with cancer, whilst tubercle fastens upon the mucous membrane of the body of the womb, and generally stops short at the internal orifice. The epididymis becomes primarily and essentially tuberculous; the testis, cancerous. In the lungs the upper section is peculiarly obnoxious to tubercle, whilst cancer occurs at every part of the lung-parenchyma. In fine, cancer and tubercle possess a different import in the most various organs according as the one or the other is primitive or secondary. Thus, cancer of the liver is not rarely a primitive, tuberculosis of this organ almost invariably a secondary affection, if not a mere participation of general tuberculosis.

3. *Typhus and Tuberculosis.*—Typhus associates itself with tuberculosis only under the influence of very intense epidemics; in other words, it very seldom attacks tuberculous individuals. On the other hand, a fibrino-croupous tubercle-crasis developes itself not unfrequently in the sequel to typhus, and with it local inflammation with fibrino-croupous exsudation of a tuberculous nature. This occurs in the shape of inflammations of the lungs, and also of such follicles of the ileum as have escaped the typhous process. This determines, from the softening of the tubercle around the typhous loss of substance, a combination of the so-called typhous with the tuberculous intestinal ulcer.

This tuberculosis in the sequel to typhus is without doubt based upon the not unfrequent conversion of the typhous to the fibrino-croupous crasis. The conversion takes place at different periods, but frequently at a very early stage of the retrogressive typhous process.

There is a similar relation of tuberculosis to the *acute exanthemata;* especially to scarlatina and measles. The tuberculosis following them is, for the most part, fibrino-croupous, and dependent upon a similar conversion of the exanthematous crasis.

4. *Intermittent Fever and Tuberculosis.*—The experience of foreigners places their incompatibility with each other beyond any doubt.

5. *Bronchocele and Tuberculosis.* — Although within the range of our own observation a moderate degree of sporadic goître has not seemed necessarily to possess an exclusive relation towards tubercle, the observations of foreigners as to the exclusive relation of *endemic* goître to tuberculosis merit, nevertheless, to be noticed here. Apart from the affinity in the structural relations of the enlarged thyroid gland, goître presents, in the out-pouring of colloid, important points of analogy with cysts, sarcomata, and cancers, in which colloid often constitutes an essential ingredient. It would seem that, in endemic goître, it is not the mechanical hinderance to respiration that occasions *consecutively*, but an anomaly of the crasis connected with the secretion of colloid in the thyroid gland, that determines *primitively* the exclusion of tuberculosis. (See *Colloid.*) This is betokened by the alienation of the general habit contracted with goître, and still more by the fact that, in districts where goître is endemic, tuberculosis does not occur, even in individuals unaffected with the prevailing deformity.

6. *Rickets and Tuberculosis* do not readily combine. Nay, rachitic deformity and coarctation of the thorax are scarcely ever found complicated with tuberculosis. It is, as yet, undecided whether, or what degree of, exclusiveness towards tubercle absolutely belongs to rickets; and, again, whether the latter owe not its immunity to a consecutive disproportion of its own creation, namely, the deformity—the narrowing—of the thorax.

7. Even the *arterial disease upon which spontaneous aneurism depends,* and which consists in the endogenous exsudation and stratification of a fibrinous substance upon the internal blood-vessel membrane (see *abnormal conditions of the arteries*) is, in its more highly developed grades, very rarely associated with tuberculosis. The immunity is, perhaps, based upon an exhaustion of the materials for tubercle, due to the deposition of

a solidifying blastema out of arterial blood. A more decided immunity is brought about by aneurisms, or by a single extensive aneurism, in the proximity of the heart, involving the endogenous coagulation of great fibrinous masses, and a consequent hydrœmia through defibrination of the blood.

8. The relation to tubercle of *venosity* (that is, an habitual preponderance of venous blood in the system) and of *cyanosis*, as resulting from mechanical hinderance at the centres of the organs of circulation and of respiration, is of paramount interest and even of great practical importance. The remarkable exemption from tubercle brought about by these conditions induces us to set forth the relevant facts, as nearly as may be, in their natural order. They determine the *venous* constitution in various ways, generally conforming in this, that they prevent the arterializing of a sufficiency of blood; whilst they engender cyanosis by hindering the return of blood to the right chambers of the heart, the said blood being arrested in the veins, and consequently in the capillaries generally. The relevant facts, ranged in a two-fold series, according as the venous habit and cyanosis are dependent upon the heart or the lungs, are as follows:

(*a*.) The first place is due to the fact, confirmed by daily experience and convenient as a starting-point for the ensuing considerations; namely, that persons labouring under enlargement (dilatation, hypertrophy, and their complications) of the heart, whether primary or superinduced by mechanical obstruction at its orifices, do *not* contract tuberculosis.

(*b*.) Nor does tuberculosis co-exist with such congenital vices of formation in the heart or the great arterial trunks [absence, insufficiency, coarctation of either, persistence of ductus arteriosus, &c.] which, with their complications, result in *venosity* and cyanosis, and, as the anatomical measure of their significance, in augmented volume of the heart.

(*c*.) Next in the series we have to mention the immunity afforded by many acquired anomalies of arterial trunks, which resemble congenital vices of formation, such as coarctation from compression, obstruction, obliteration, or again by large aneurisms in the vicinity of the heart. Apart from what has already been said on this point, the immunity is due to the mechanical impediment which the overpowering blood-column in the dilated aortal trunk opposes *directly* to the emptying of the left ventricle, and indirectly to the influx of venous blood into the right heart.

The same immunity is attained in venosity and cyanosis owing to hinderance to the pulmonary circulation; more especially where the impediment reveals its serious character by a dilatation of the right heart.

We may here further adduce:

(*d.*) The observation that the *increased density* of the lungs produced by *coarctation of the thoracic spaces,* in higher grades of lateral curvature of the spine, or in the rickety chicken-breast, excludes tuberculosis. Nay! it is an important fact that, with the establishment of a deformity of the spine in the shape of gibbosity, even when owing to tuberculous caries of the vertebræ, the tubercle-crasis is for ever rooted out in consequence of the narrowing of the thoracic spaces.

(*e.*) The fact that the compression exercised by pleural effusion, and a consecutive, abiding increase of compactness of the one lung, as denoted by a sinking in of the thorax, in like manner extinguishes the tendency to tuberculosis. This effect is the more surely produced, the greater the mechanical obstruction, and the consequent disproportion between the blood-mass and the lung-capillaries pervious to it; and the less competent the other (vicariating) lung is to carry on the function of arterialization.

(*f.*) The fact that pregnancy arrests the progress of an established tuberculosis; or, as we would correct and extend this proposition, the fact that advanced pregnancy not only arrests a tuberculosis already in being, but also obviates the formation of tuberculosis generally. It is the effect of that embarrassment of the thoracic spaces, and of that resulting condensation of the lung-parenchyma occasioned by upward pressure from the abdomen; in other words, it is based upon a *venosity* brought about by mechanical means. It is probably for similar reasons that the placenta very rarely,—the fœtus perhaps never,—becomes tuberculous.

This relation derives further interest from the rapidity with which, after child-birth, that is, after removal of the conditions which prevailed during *advanced* pregnancy, fibrin-crases with their respective exsudatory processes, and amongst them the tubercle-crasis and tubercle-deposits, take place, more particularly through the medium of inflammatory stasis.

(*g.*) To the same class is to be referred the immunity from tubercle arising from every enlargement of the abdominal space, and the consequent narrowing of the thoracic cavity. The exemption allotted to patients afflicted with vast ovarian cystoids probably partakes of this nature.

(*h.*) Again, the fact that even congenital smallness of the pleural sacs, paired with primitive smallness of the lungs, and, as it mostly is, with an inverse ratio of the development of the abdomen and its viscera, serves as a protection against tuberculosis;

(*i.*) That in the earliest childhood (with closed fœtal passages), owing to a condensed state of the lungs caused by predominant abdomen, tuberculosis occurs, if at all, very rarely.

(*k.*) The exemption apportioned to those who labour under chronic catarrh, under vesicular emphysema of the lungs, or under bronchial dilatation, was recognised even by Laennec. The empirical recognition of this relation has even led to attempts to cure tuberculosis by the forcible production of those conditions. The real preservative point was, however, overlooked, both here and in another mode of cure aimed at by others, namely, that of closing cavities in the lungs by forcible compression of the thorax. The protective and curative impulse consists, even here, in *venosity*. And this venosity is a consequence of the destroyed function, the collapse and eventual wasting of numerous pulmonary lobules, through obstruction of their bronchia with muco-purulent secretion; in bronchial dilatation, through the concomitant obliteration of considerable portions of the lung; in emphysema, through lost contractility of the pulmonary texture for exspiration, and consequently embarrassed respiration, more especially, however through the destruction of extensive ranges of the lung-capillaries.

(*l.*) It will be readily understood that the dropsical crasis, especially when resulting from *venosity*, excludes tubercle.

It will now become necessary to inquire how certain exceptional cases are to be explained. Individual cases of the kind are represented in tubercle associated with cancer, or with *venosity* mechanically brought about.

(1.) The conditions mentioned as excluding tubercle, operate thus only in so far as the latter is based upon a hyperinotic crasis—*an excess of fibrin.* This does not, however, prevent the small fund of fibrin accompanying those conditions from being, under favorable circumstances, expended upon tubercle formation, which then becomes localized in a process of exsudation.

(2.) The tubercle may be the product of a *local inflammation,* in which the fibrin becomes tuberculous.

(3.) The entire mass of fibrin may suffer a morbid change, effecting, as intercurrent disease, a consecutive tubercle crasis, which, becoming exhausted by a corresponding exsudation, again gives way to the original crasis.

It is thus that genuine tubercle, when concurrent with cancer, may be interpreted; and this the more readily, that true hyperinoses and fibrinous exsudates not unfrequently do coexist with cancer. The tubercle may be merely local, and the cancer no less so. It may, however, be local, and yet the cancer be a general disease. Or, again, it may be the product of an intercurrent primitive tubercle-crasis, or of a consecutive one derived from a local process, and coordinate with those hyperinoses and fibrin-exsudations which not rarely supervene upon inflamed and ulcerated cancer, reflecting a secondary crasis.

(4.) As to the exemption afforded by *venosity,* there is no doubt that, to render it complete, a high degree of the latter is requisite. Since, however, we possess no scale whereby to ascertain directly the grade of a protective crasis, and to illustrate the exceptions, we must inquire whether it be not possible to arrive indirectly and approximatively at this recognition. In the absence of such a scale, certain anatomical changes must serve as the measure, so to speak, of the anomaly. They consist in the degree of heart affection (dilatation) present, this furnishing an available criterion for the amount of the impediment to the circulation, and therefore for the grade of the venosity. This approximative index with the aforesaid inferences, will be especially applicable where the precise extent of the impediment is not to be immediately summed up from anatomical data, as in lung affections, like catarrh and bronchial dilatation, emphysema, and preternatural density of the lungs.

We attach importance to this relation of tubercle to the venosity resulting from mechanical impediments in the heart and lungs,—as affording not alone proof of the fibrin-crasis being the foundation of tubercle but also valuable indications for medical treatment.

We have now to consider the relative occurrence of tubercle in the different organs and textures, and its peculiar processes of repair.

It will be expedient, however, as a preliminary point, to determine what is signified by scrofula,—what is the distinction—if there be any—between scrofulous and tuberculous substance.

For our own part we hold *tubercle* and *scrofula* to be *identical* —tuberculosis and scrophulosis to be one and the same disease; and this upon the following grounds, namely:

(*a.*) One and the same elementary composition, both anatomical, and, so far as investigation has gone, chemical also. This applies with especial force to scrofulous substance, as compared with *yellow* tubercle.

(*b.*) Both are subject to the same metamorphoses, namely, softening and cretefaction.

(*c.*) The tuberculous and the scrofulous ulcer are identical both in the same, and in different organs; for example, the scrofulous skin- and the tuberculous intestine-ulcer. The same identity attaches to their cicatrix.

(*d.*) Both frequently coexist in the same organ, sometimes without, sometimes with, the appearances of inflammation.

The truth is, that the yellow tubercle is commonly called "scrofulous substance," more especially when it occurs in largish masses, and affects in the usual way the glands—the lymphatic glands—in children. Thus the same substance concurrently affecting the lungs and the bronchial glands is denominated, in the one instance tubercle, in the other scrofula.

A scale of the frequency of tubercle in the various textures and organs, offers but limited points of interest. According to our experience, it would present in adults something like the following series, namely:

Lungs.
Intestinal canal.

Lymphatic glands, more particularly the abdominal and bronchial.

Larynx.

Serous membranes, especially the peritoneal and pleural.

Pia mater.

Brain.

Spleen.

Kidneys.

Liver.

Bones and periosteum.

Uterus and tubes.

Testicles, with prostate gland and seminal vesicles.

Spinal cord.

Striated muscles.

For children this scale does not answer completely. In them the lymphatic glands, together with the spleen, would take the lead, followed by the lungs with the bronchial mucous membrane, the brain, the serous membranes, &c.

The ensuing remarks appear to us well deserving of attention, as affording evidence of the imperfection of *any* summary scale of frequency.

(1.) At every point where capillaries occur, there may be tubercle. Epidermid formations and cartilage are therefore alone exempt from tuberculosis.

There are, however, vascularized organs in which tubercle very rarely, if ever, occurs; such are the salivary glands, the ovaries, the internal blood-vessel-membrane, the œsophagus, the vagina.

Even vascularized new-growths may become the seat of tubercle.

(2.) If, which is most important, we consider tuberculoses individually, according to their primitive or to their secondary appearance, an entirely different scale is set up. The lungs and lymphatic glands, it is true, retain their uppermost rank, but are immediately followed by tuberculoses, which stand very low in the foregoing scale, namely, of the urinary system, of the female sexual mucous membrane, of the bones, of the testicles with the prostate gland and the seminal vesicles. Meanwhile tuberculoses of the intestine, of the larynx and trachea, of the serous membranes, of the spleen and liver, take a very subordinate position in the new scale, seeing that they seldom, if ever, become the primary seat of tubercle.

(3.) Accordingly, certain tuberculoses which in the first scale occupy a high place, possess but a very subordinate nosological import. They are seldom, if ever, primitive, but almost always secondary, dependent upon other tuberculoses, often, indeed, only participant in general tuberculosis. The liver, spleen, kidneys, nay, in many cases the lymphatic glands, stand in this relation to tubercle.

(4.) Tuberculosis almost invariably attacks several determinate organs concurrently, at the outset or at a very early period. Of this communion we have examples, not only in the joint tuberculosis of lymphatic glands and of the implicated organs, but also in that of the brain and of the lymphatic glands; of the testis, prostate gland, seminal vesicles, and of the urinary organs; of the spleen and supra-renal gland, and of the lymphatic glands; of uterine and tubal, and of peritoneal; of pulmonary, and of intestinal, or of laryngeal tubercle.

(5.) Secondary tuberculoses have a sort of ground-work or starting point in certain pre-existing tuberculoses. In other words, secondary tuberculoses accede to already existing ones according to a tolerably constant rule. Thus, tuberculosis of the lungs or lymphatic glands offers for all such secondary tuberculoses, a general point de départ, whilst, on the other hand, it commonly associates itself to most other tuberculoses. Tuberculosis of the serous membranes accompanies that of the implicated parenchymata; tuberculosis of the urinary system, that of the genital apparatus in the male. (See 'Tuberculosis,' vol. iv.)

(6.) Again, the mode of production of tubercle varies in the different organs. Thus, upon serous membranes and in bone, tubercle is, for the most part,—upon mucous membranes, very frequently,—in lymphatic glands and in the brain, not unfrequently the product of inflammation.

(7.) In fine, it is worthy of note that in every organ tubercle, unless thrown out with much violence, has its almost invariable, and readily demonstrable point of incipiency. In the lungs it is at the apex, the upper third of the superior lobes; in the pia mater, at the part investing the base of the brain within the common groove, running from the chiasma to the pons

Varolii and the medulla oblongata or about the fossæ sylvii; in the brain itself, in and about the gray substance; in bones, in the spongy bones or parts of bones; in intestinal mucous membranes, in that of the inferior ileum; in the laryngeal mucous membrane, at the portion covering the transversus glottidis muscle; in the testicle, in the epididymis; in the female sexual apparatus, in the mucous membrane of the tubes and uterine fundus—that the deposition of tubercle first commences and concentrates itself.

(8.) Again, there are a few marked limitations set to the advance of spreading tubercle. For example, tubercle of the larynx never extends to the pharynx; uterine tubercle hardly ever passes beyond the internal orifice, so that the cervix uteri and the vagina remain exempt.

Tuberculosis very commonly proves fatal, if locally, by impeded function, by palsy of the affected organ, in consequence either of the extensive, acute deposition of tubercle into its texture, or else of the ulcerous destruction of the latter in the process of so-called tuberculous phthisis. Or the tuberculosis may, as a general disease, destroy life through impoverishment of the blood, through hydræmia or the serous crasis, an issue vastly favoured where the tubercle is copiously and at the same time rapidly thrown out, and where local tuberculosis in important organs hinders the reproduction of blood.

The cure of tubercle may take place in various ways. Each of the metamorphoses of tubercle may become invested with the character of a healing process. Still, neither the decadence of tubercle, nor its ejection through the medium of ulceration, as local healing processes, are fraught with any value for the individual, unless accompanied by the extinction of the fundamental, tubercle-producing crasis.

The cure of tuberculosis as a general disease—as tubercle-dyscrasis—takes place now and then obviously through the intervention of some of the processes and conditions already adverted to as excluding tubercle; at other times, through influences entirely occult.

A question connected with the local healing process of tubercle here suggests itself, namely, as to the absorption of crude tubercle? The resorption of tubercle as formerly believed in, was probably first repudiated by Laënnec, and after him by most pathologists; and although valid grounds can hardly be alleged for its impossibility, neither has it ever been proved by direct evidence, nor is it at all within the compass of likelihood.

The *obsolescence*, the cornification of gray tubercle, represents incontestably its readiest process of involution. As a direct extinction of the tubercle, it would afford the completest cure, did it not concern a growth which would fail to become destructive if it abided in its primitive crude condition.

Of the two other metamorphoses affecting yellow tubercle, cretefaction of what has undergone softening unquestionably presents the most desirable process of repair, as will become evident from the following remarks concerning the other metamorphosis, considered as a healing process, or as the basis of one.

The softening of tubercle cannot of itself serve for a reparatory process. The *elimination* of softened tubercle through the instrumentality of ulceration in its vicinity, can alone pass current for such.

But, taking into account—

(*a.*) That it can only be brought about by ulcerous destruction of the textures.

(*b.*) That, although the aim of this ulcerous process be to heal, it may, when the tubercles are numerous, readily induce exhaustion.

(*c.*) That the attendant inflammation—the general disease being unextinguished—of itself determines tuberculous products, thus extending, without limit, the ulcerous consumption of the textures.

(*d.*) That even under favorable crasial conditions, an infection of the blood is possible in tuberculous ulcers (cavities).

Taking, we say, all these circumstances into account, this curative process must be regarded as widely subordinate to that of cretefaction, to which it stands in nearly the same relation as the removal from the body of a foreign substance by a debilitating ulceration, to the same substance being rendered innocuous by incapsulation.

The healing of a tuberculous ulcer or cavity,—of tuberculous ulceration,—can therefore only take place provided the accompanying inflammation, owing to extinction of the tuberculous crasis, ceases to deposit fresh tuberculous matter, and determines organizable products instead. The loss of substance is made up for by new-formed shrivelling scar-texture. Where the tubercle has not been completely eliminated in the phthisical process, the residue may become isolated by a pap-like inspissation and eventual cretefaction.

c. ALBUMINOUS TUBERCLE. *Acute Tuberculosis.*

Under this denomination is understood a disease presenting many points, both of resemblance and of dissimilitude with the tuberculoses already discussed. It devolves upon us to investigate these analogies and differences; the former appearing to us to preponderate so far as to preclude our separating the disease from tuberculoses generally.

There is a disease which, under an acute course, and under typhoid symptoms, determines a tubercle differing in many respects from the fibrinous. It always represents solid, mostly poppy seed-, rarely, if ever, millet seed-sized, sometimes limpid, softish, glutinous, gray granulations, either of vesicle-like or of a dull transparency, often only cognizable under a favorable incidence of light; at other times, although far less frequently, opaque, whitish, or whitish yellow.

On a closer inspection, this tubercle appears marked by cell-formation. It is found to contain—

(*a*.) The ordinary nucleated exsudate cell in considerable numbers.

(*b*.) Cells with two or three nuclei.

(*c*.) Cells with filial cell-formation.

(*d*.) A structureless soft basement connecting these elements.

It exsudes always in great abundance under the symptoms of hyperæmia, in scattered granulations, uniformly distributed through the parenchyma of the affected organ, and either all at once, or at intervals rapidly succeeding each other. A manifest equality of size and character is observable in all those deposited simultaneously, or during the same attack. With it there is always effused a grayish, sero-albuminous semi-gelatinous humour, with which the diseased textures become infiltrated.

This tuberculous deposition affects not only entire organs or large sections of organs and of textures, but commonly several organs and textures simultaneously or in rapid succession; a single one, however, generally operating as the main point of concentration. Its seat is in the lungs, the pia mater, especially at the base of the brain, the spleen, the serous membranes, especially the peritoneum.

This tuberculosis is only in rare instances the primitive one. For the most part, it is based, so to speak, upon a precursory fibrin tuberculosis of the lungs or lymphatic glands. In these cases its point of concentration is generally either the organ previously affected, or some structure standing in immediate relation with it.

The disease proves fatal through palsy of affected organs essential to life, or else through dyscrasial influence.

This tubercle is subject to no metamorphosis.

The drycrasial character of the blood is manifest, and closely assimilates to the exanthematous crasis. (See 'Crases.') In accordance with it are the livid coloration of the common integument in the dead subject, the dark coloration of the muscles, the general appearance of flabbiness, the serous infiltration of the parenchymata.

In this description of the disease we recognise the albuminous crasis, and a product which, in its subordinate coagulability, its soft, gluey character, its cell-development, gives evidence of its *albuminous* nature.

This tubercle renders it probable that albumen, without previous conversion to fibrin, may acquire a considerable amount of coagulability, and become tuberculous; nay, that where the opaque yellowish or whitish-yellow acute tubercle does not form upon a basis of croupous fibrin, even albumen may, without conversion into fibrin, acquire the croupous character.

In this description we recognise not only the distinctions, but also the analogies between the fibrinous and the albuminous tubercle. These analogies stand forward the more prominently if we recall to mind the fibrin tubercle of acute production.

Such analogies, apart from the resemblance in outward form of the two heterologous deposits, that is, the tubercle form; apart from the uniform size of the granulations thrown out at the same period of exsudation; and apart from their equable dissemination through the parenchymata; are as follows:

(*a.*) Both are rarely the primitive tuberculoses in an organism.

(*b.*) Both are thrown out under manifestations of hyperæmia.

(*c.*) With both there is effused, as a sort of vehicle for the coagulable portion of the entire exsudation, a serous, sero-albuminous fluid.

(*d.*) Both affect the same organs and sections of organs.

(*e.*) The albuminous tubercle bears the same relation towards other diseases as the fibrinous.

(*f.*) In by no means rare instances, a step-like transition from the fibrinous to the albuminous tubercle is incontestable. Upon the groundwork of a fibrin-tuberculosis, which has undergone frequent phases of phthisis, there exsudes, with augmented dyscrasis, in the lungs more particularly, a tubercle which, with every fresh act of exsudation occurring in rapid succession, becomes softer and poorer in fibrin, until ultimately reduced to a soft, semi-fluid, albuminous tubercle,—a consummated acute tuberculosis.

(*g.*) Occasionally we discover, especially in the texture of the pia mater at the base of the brain, an exsudate consisting of albuminous tubercle and tuberculizing croupous fibrin, a primitive combination of the two products.

(*h.*) Not only does albumen enter into the composition of of fibrin tubercle, but a certain amount of fibrin modifies that of the albuminous tubercle. A complete exclusion of the one or the other is hardly conceivable, and it is only the predominance of the one or of the other that characterises the product. Between the extremes of fibrinous and of albuminous tubercle there exist numerous middle and transition forms.

ALBUMINOUS CRUDE BLASTEMATA.

Under this head we shall discuss certain products in their nature probably albuminous, and essentially distinguished from other albuminous blastemata by their persistence in the condition of crudity. Owing to this persistence, as also to their being founded in a dyscrasial element, we rank them along with tubercle, with which, moreover, they occur not unfrequently in consecutive alliance.

They are, for the most part, solidified blastemata, resembling to the naked eye a translucent coagulated albumen. Now and then, however, they are opaque, and of a turbid whiteness. They consist of an amorphous, glebous, transparent basement, and of nucleus formations.

They occur in certain parenchymata in the shape of infiltration—very rarely in that of a collection of roundish nodules from the size of a hemp-seed to that of a pea. The liver, the spleen, and the kidneys are known to become affected with these infiltrations, which give to the surface of the organ a brawn-like aspect with a transparent margin, frequently representing a spurious hypertrophy of the organs named. (See 'Hypertrophy.') Whether these blastemata occur in one, or in several, or in all of those organs, they mostly occasion considerable enlargement thereof, and at the same time a notable change in their consistency, the parenchyma becoming compact and of doughy brittleness.

The out-throwing of these blastemata occurs in an insensible manner.

In point of fact, they comprise that partly more or less solidifying, whitish, partly viscidly fluid blastema effused into the parenchyma of the kidney in Bright's disease, particularly in certain of its chronic forms.

As may be inferred from the above, and as experience amply confirms, these blastemata never occur but in connection with high grades of general dyscrasial disease; such, for example, as rhachitis, mercurial cachexia, inveterate syphilis, ague-cachexia, and especially certain tuberculoses.

The deposition of these blastemata is, therefore, never a local affection, but invariably indicative of an anomaly of general nutrition. It is clearly dependent upon dyscrasis, which may consist in an excess of albumen in the blood, and be either primitive or secondary, as in the tuberculosis resulting from the exhaustion of fibrin. The consequence of a copious and extensive secretion of these blastemata is the eventual exhaustion of albumen, and a watery condition of the blood, [hydræmia,] inductive of dropsy, anæmia, &c.

These blastemata usually abide altogether, and throughout, in their primitive condition. Occasionally, however, there is observable, at certain spots, a transformation of their mass into molecular fat. They become opaque; of a whitish dulness; friable. This is especially the case in the liver and kidneys, and it is not improbable that cera-lardaceous infiltration of the liver is the result of a progressive, diffused conversion of this albuminous blastema.

II.—UNORGANIZED NEW-GROWTHS.

A.—OF UNORGANIZED NEW-GROWTHS IN GENERAL.

These lack both the internal order and the definite forms which characterise organized new-growths, and their development comes under the dominion of chemical laws. Between the *rudiments* of what *is*, and what is *not* organized, there is no distinction in point of form. In a chemical sense non-organized growths are composed both of unorganized and of organized substances, either singly or conjointly, and it is even common enough for a new-growth to be made up through the mechanical blending or interlacing of organized with unorganized materials. All these considerations taken together preclude any marked discrimination between the two.

There are, upon the one side, undoubtedly new-growths representing perfect unorganized formations, for example, certain concrements. On the other side, however, non-organized new-growths originate under conditions and forms which have induced us, notwithstanding their unorganized nature, to discuss them along with the organized new-growths. We may instance the forthissuing of lime-salts—as cretefaction, ossification, incrustration; of the free fats; of colloid; of tubercle.

The material for non-organized new-growths in general, is contained both in the textures, and in fluid and solid blastemata; the material for a special order of non-organized new-growths, in the proper fluids of secretion. Its nature varies considerably. It consists of protein substances, certain gluten-substances, horn-substance, fats, pigments, acids, salts. In a more extended sense, even the various gases and fluids occurring in textures, or in the cavities of the body or of organs, the fluid of genuine dropsy for instance, belong to the class.

Without for the present taking these last into the account, we have to observe with reference to unorganized new-growths :

The elementary forms are the amorphous, the glebous, the laminate, the granular (down to the finest molecule or point-mass), the crystalline. Certain substances possess a determinate form, dependent, however, for the most part, upon their peculiarity of composition, upon the conditions under which they become severed from their primitive combinations, and upon their mode of development. Thus protein substances occur, both structureless, and in a glebous or a molecular form.

These materials constitute secondary formations, either alone or with the intervention of a bond-medium, for example, mucus. This is often furnished, together with the external moulding or form of the new-growth, by the glutinous basis of a texture; for example, in the so-termed ossification of a fibrous tumour. Both in form and size they manifest great variety, not readily susceptible, however, of generalization. We allude more particularly to calculous concretions! In consistency they are in various degrees soft or firm.

Above all, their chemical composition varies greatly. As regards concretions and calculi, these readily divide into two groups, namely, into such as form out of fluids of secretion, and consist of the respective components of those fluids, and into such as become developed out of blastemata and textures. These last have a composition corresponding with their base, and very commonly consisting of phosphate and carbonate of lime, and of magnesia.

Respecting the origin—the mode of production—of non-organized new-growths, it may be stated generally:

1. They are exsudates or secretions in a primitive form of non-organization, as exemplified in crude fibrin, and encysted colloid and fats.

2. They are the result of various transformations of such products. To this order belong:

(*a*) Formations arising out of the conversion of exsuded and secreted protein materials into glutinous, into horny substance, into fat; for example, the conversion of fibrin and albumen to colloid, to horny substance, to fat in the molecular or crystalline form.

(*b*) Formations arising out of a process imitating ossification in fluid or solidified, unorganized or textural bases; a liberation of lime-salts (phosphate and carbonate) out of their primitive.

combinations, as cretefaction, ossification, lime-incrustation, concretion. (See 'Bone Formation.')

(*c*) Formations brought about by a more palpable deposition of all, or only of certain, components of a fluid in which they are held in solution or suspension. They are most especially prone to form in secreted fluids, and either consist purely of specific ingredients proper to them, or occur blended with other elements. *They constitute calculous concretions.* The cause of their separation is manifold. It may be that the fluid has become more concentrated, for example, by loss of water, their solvent medium, through exosmosis (resorption) or more especially by evaporation. Again, we may mention, besides the precipitation from fluids of certain specific components, the inspissation and exsiccation of secreted and exsuded fluids in their totality; for instance, of mucus, of ear-wax, of the smegma præputii, of the bile, of exsudate, of pus, &c. Or else it is a consequence of a chemical conversion of the fluid,—of the solvent, or of the dissolved substance. For instance, the free acid of normal urine retains the phosphatic earths in solution; when, however, the urine is rendered alkaline, be it by the presence of mucus or exsudate, or by conversion of the urea into carbonate of ammonia, the phosphatic earths become precipitated. If the lithates present in the urine become decomposed by an excess of acid in the urine, the lithic acid, as the less soluble, is thrown down. The soluble phosphate of magnesia present in almost all the fluids becomes precipitated, the moment that it enters into a combination with ammonia, to ammonio-phosphate of magnesia.

Unorganized new-growths possess sometimes a local, sometimes a general import. Thus, urinary calculi may be the result either of mere local contingencies, or of various anomalies of general nutrition, that is, of a dyscrasial process.

B.—OF UNORGANIZED NEW-GROWTHS IN PARTICULAR.

We have here, in the first place, to bring forward and to examine in detail the substances which constitute new-growths.

1. *Protein substances.*—The primitive form in which these emerge from their solutions, is that of a structureless or

glebous mass, in various degrees of coagulation, and that of an elementary granule down to a pulverulent point-mass. The reason for their appearance in these forms, that is, the reason for the general coagulability, and *a fortiori* for their specialities of form and coagulation is quite obscure. That which spontaneously undergoes rapid and firm coagulation passes current for fibrin; that which coagulates more slowly and less perfectly under a manifest progressive change in the medium of solution, for albumen. The molecular form appertains in particular to the higher grades of oxydation of the protein substances, (croupous aud pyin-holding fibrin). Since the influences which produce the coagulation and precipitation of albumen in experiments, do not presumably take place within the organism, the discovery of the modifications suffered by albumen through the agency of water, acetic acid, and the like, is highly deserving of attention.

The protein deposits are insoluble in ether and in mineral acids. By acetic acids they are rendered translucent, and ultimately dissolved. By caustic potash and fuming hydrochloric acid they are slowly dissolved—by the latter with a lilac tint. An aqueous solution of iodine colours them yellow.

The *glutinous* and *horny* substances emerging out of the protein-substances are amorphous, or have a glebous or a stratiform, elementary composition. In their physical properties they approximate, more or less, according to their grade of perfection, to gluten and to urea; in their chemical reaction, to various modifications of gluten (gluten, chondrin, pyin, &c.), and of urea.

2. *Fats.*—Their elementary form of occurrence is that of *drops*, or of an amorphous solidification; of granules; of crystals. Little is known concerning the nature of fats originating thus, more especially of those assuming the form of granule (elementary granules, both free and incelled, discrete and aggregate), or developed out of blastemata and textures through conversion of protein-substances, and probably even of gluten.

The fats cognizable by their form and chemical relations are:

(*a.*) *Elain.*—It occurs in variously-sized drops, both free and incelled. In this form it is usually set free out of emulsion-like compounds; in the form of *elaic acid,* out of saponaceous compounds, or out of combinations with other fats, for example, in exsudates, in medullary carcinoma. It frequently represents, an effusion of the contents of fat-cells, consequent upon gangrenous or ulcerous destruction. The drops resist the action of water and of acids, but dissolve on being boiled with potash, and still more readily in ether or heated alcohol.

(*b.*) *Margarin and margaric acid.*—These occur in microscopic needle-crystals, for the most part aggregated in stellate groups or bundles. In this shape the margarin emerges, after the body has become cooled, from its solution in elain, either within the fat-cells, or without. The crystals of margaric acid, soluble in concentrated, heated alcohol, are, according to Vogel, probably a product of decomposition, wrought out of the margarin of the fat, it may be, by a free acid, so often developed in gangrene.

(*c.*) *Cholesterin.*—When cognizable as such, it occurs in tabular crystals, representing rhombic planes. Many, however, of the aforesaid fat granules are likewise cholesterin. It almost always occurs along with other fats, and often very copiously; for instance, in gall-stones, in the atheroma of arteries, in encysted tumours. The fact of cholesterin so frequently occurring in fluid and solidified protein substances during their disintegration,—as in exsudates, in tubercle, in stratiform coagula upon the inner coat of arteries, renders it probable that, like other fats, it is the product of a decomposition of the elements of those substances. This seems to us more probable than that it exists preformed in combinations which cause it to be held in solution. Its detection in the blood does not appear to us a valid objection to this. It is soluble neither in water, nor in acids, nor yet in alkaline solutions, but only in ether and heated alcohol.

(*d.*) *Stearin.*—Its occurrence is not proved with certainty, although, under certain conditions, in which fat assimilates to the suet of the wether, not quite improbable.

3. *Pigments.*

(*a.*) *Black, brown, russet-yellow pigment* (See 'Pigment'), in the shape of molecular granules; the last two occur, also, adherent to microscopic crystals of ammonio-phosphate of magnesia.

(*b.*) *Bile-pigment*, as a finely granular precipitate of a yellow-brown colour, insoluble in water and in most of the acids, —soluble in a boiling potash-solution, with a greenish-brown tint. Nitric acid destroys it, after causing it to pass through phases first of green, then of blue, and lastly of red coloration.

4. *Lithic acid and lithates.*

(*a.*) *Lithic acid.*—The fundamental type of its crystals is the rhomboid prism, which, however, often appears cut down to a rhombic plane. The crystals, frequently seen grouped into rosettes (Simon and Vogel), are difficult of solution in water, insoluble in acids, alcohol, and ether. Potash causes their gradual solution. They occur in the urine.

(*b.*) *Lithate of ammonia*, as a finely granular precipitate, coloured of a dingy yellow, yellow-red, russet, rose-tint; difficult of solution in cold water; less so in hot. The effect of acids is to isolate the lithic acid, which, under the microscope, is then seen to develop its crystals.

5. *Lime-salts.*

(*a.*) *Basic phosphate of lime*, as a gelatino-granular mass, soluble in acids. It occurs both in fluids and in solidified formations, in a soluble combination of protein-substances, with gluten, out of which it separates—especially in the shape of cretefaction and ossification—in the form of molecule.

(*b.*) *Carbonate of lime*, in the shape of granular deposition,—in the cell-incrustation, of stratefaction—either alone or in union with the foregoing substance. Soluble in acids, with effervescence.

(*c.*) *Oxalate of lime*, in octohedral crystals, sometimes remarkably minute; insoluble in water, alcohol, ether, acetic acid; soluble in hydrochloric acid. Found in the urine.

6. *Ammonio-phosphate of magnesia*, in crystals of various shapes. When rapidly formed, they cluster together in stellate groups of needle-shaped crystals, or represent denticulate, leaf-like forms. When slowly developed, they constitute trilateral prisms, in which both angles corresponding to the same lateral-edge are truncated. The crystals are readily soluble in acids—even in acetic acid. The occurrence of this salt is extremely frequent. Wherever a development of ammonia takes place, the wide dissemination of phosphate

of magnesia determines the formation of the insoluble triple phosphate.

7. *Sulphuret of iron*, in molecular granules, soluble in acids and precipitable out of these by means of sulphuret of soda.

Such are the principal and the better known substances which, independently of, or in combination with, others, compose the bulky unorganized formations, as so-called concretions or concrements. We shall treat of these generally; dividing them into two great series, namely:

1. Into such as are essentially protein-substances; or into such as consist of gluten or horn-like substance, of fat, and, lastly, of the phosphates and carbonates of lime and magnesia; it matters not whether the latter be directly thrown out as such, or whether they have, as usual, become obviously developed out of the former, that is out of the protein-substances.

2. Into such as have comparatively a varied composition, and are marked by the specific substances which they contain.

FIRST SERIES.

(*a.*) *Protein concretions*, as coagulations within the blood-vessels—*vegetations;* as exsudates in parenchymata, upon membranous surfaces; as free bodies in serous cavities, tubercle, &c.

(*b.*) *Accumulations of gluten-like, colloid substance*, commonly within cyst-spaces; and *cornified protein concretions*, for example, of the valve-vegetations in the heart.

(*c.*) *Fat*, as the cyst-contenta; or as accumulations within serous cavities; in parenchymata, in the shape of spherical or irregular masses. Frequently in combination with the following, namely:

(*d.*) *Bone-earth concretion*, so-called cretefaction and ossification, as developed, not alone in the protein and gluten-holding unorganized basements already adverted to, but also in fibroid and cartilaginous textures, in a mode and form which, together with their relations to the normal ossification of bone-cartilage, we have discussed in another part of this volume. [See 'Bone Formation.']

The concretions consisting of *lithate of soda*, found in the sheaths of tendons, within capsular ligaments, even in the spongy texture of the articular terminations of bones, consti-

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The concretions consisting of *lithate of soda*, found in the sheaths of tendons, within capsular ligaments, even in the spongy texture of the articular terminations of bones, constitute an exception only as regards the nature of the substance itself.

The significance of concretions of this series differs with the organ affected; thus, concretions on the heart's valves are, perhaps, the most important of all.

SECOND SERIES.

To this series belong the concretions in and arising from fluids of secretion. We divide them into two species:

(*a.*) They result from the *precipitation of one or of several of the specific components of a secretion*, animal matter entering into their composition for the most part only in small quantity and by way of a bond medium.

They constitute the genuine stony concretions or calculi, which, when diminutive, are termed gravel or sand.

The size of calculi is, as may be inferred from what was just stated, extremely various, from that of a fine, just perceptible sand-grain, to that of a concretion filling up the largest secretory canals and reservoirs.

In smaller, solitary concretions the form is mostly the spherical;—in larger ones, it corresponds to that of the said canals and reservoirs, as in the instance of renal calculi, and is subject to much modification. Where many concretions co-exist, they acquire from reciprocal pressure and friction, the most varied, polyedrical shapes,—as in the case of urinary calculi, and especially of gall-stones. Their surface is smooth, polished, or else knobbed and uneven, stellate, thorny, rough.

Their consistence mainly depends upon their chemical composition.

They reside at large in their respective cavities, or else, filling the latter, they lie firmly impacted. Or again, they adhere as if glued or soldered at some point, through the medium of fibrinous exsudate.

Their structure varies extremely. At their nucleus they exhibit an agglomeration of an amorpho-granular precipitate. Or again, they consist of concentrical strata of the same character, or else of a crystalline precipitate; or lastly, they are altogether of crystalline fabric, as in the case of certain lithic acid calculi, but particularly of cholesterin concrements in the gall-bladder.

The first impulse to their formation is sometimes given by foreign bodies introduced from without, or by coagulate,—endogenous products. The concretions represent, in the first instance, incrustations of things in various degrees alien to their composition. Thus, for example, on the one side, a great variety of foreign bodies which have lapsed into the urinary bladder, give rise to lithic acid calculi; on the other side, inspissated bile, or bile-pigment, to cholesterin calculus in the gall-bladder.

To this category belong lithic acid calculi, salivary calculi, lacrymatory calculi, prostatic calculi, gall-stones, many intestinal concretions.

(*b.*) They are due to the ***inspissation and desiccation*** [through exosmosis or evaporation] of some fluid of secretion either within or externally to its secreting canals and cavities. Here the concrement consists of the ingredients of the secretion in their totality, including, of course, a considerable amount of so-called animal matter, and with it of organized elements. Proportionately to the degree of inspissation, the concrement is soft; or, it may be, of a stony hardness. Concretions of this kind very often become developed in cyst-like dilatations of the follicles in which the secretion accumulates and stagnates, and the inspissation of the contents of encysted tumours of new formation, applies here in its most extended sense. The physical and chemical properties are, it will be readily conceived, extremely inconstant and variable.

This group comprises concretions in the follicles of the skin, in mucous follicles, in the tonsils, in the nasal and pharyngeal cavities, upon the glans and prepuce, and certain intestinal concrements, especially those occurring in diverticula; finally, the inspissations of colloid, and of other cyst-contents.

CHAPTER X.

ANOMALIES OF CONTENTS.

In this chapter we have to treat of:

A. *Pneumatoses* and *Dropsy* which we have already adverted to as non-organized new formations.

B. *Foreign substances* introduced into the body.

C. *Parasites,* that is animals, and vegetable growths, occurring in and upon the living body. We give them a place in this chapter because, according to the researches of modern science they are to be numbered amongst the things that are received into the organism from without.

A. PNEUMATOSES AND DROPSY.

1. *Pneumatoses,*—the accumulation of various gases has been observed as emphysema, both within textures, and more particularly in almost every cavity of the body and of its organs. The scale of frequency varies, indeed, according to the nature of the gas, and to its mode of origin. There are, however, organs in which gas-accumulations of every kind are extremely common; and again, others in which a development and accumulation of gas are under all circumstances very rare.

The modes in which gas-accumulations originate resolve themselves generally, into the following:

(*a.*) The gas accumulated in the texture or in the cavities of the body or of organs, is *atmospheric air* which has penetrated from without. This applies to most kinds of emphysema, and of gas-accumulations in the pleural sac, partly to those in the stomach, perhaps also to the rare instances of gaseous collections in the uterus, and in the urinary bladder; lastly, to the presence of gas in the blood after the lesion of veins, particularly those of the neck. Most examples of interstitial emphysema and of pneumothorax are the result of lesions of continuity, through either wounds or ulceration in the bronchial passages or in the lungs.

By tarrying in preternatural localities, the atmospheric air suffers a change similar to what it undergoes in the lungs, its oxygen becoming exchanged for carbonic acid, with the superaddition of aqueous vapour.

(*b.*) *The gases are products of decomposition.*—To this category are to be reckoned, besides those gas-accumulations arising out of putrefaction after death,

a. Gas-development out of the blood-mass, from putrid decomposition of the latter; out of blood perishing through absolute stasis; finally, out of decaying normal textures or morbid products, for example, sloughing cancers, or exsudates undergoing decomposition.

β. Gas-development in the stomach and intestines, the details of which concern special anatomy.

2. *Dropsy*, whereby we understand *genuine serous dropsy*, that is, a fluid mostly alkaline, in its purity colourless and limpid, and analogous in the quality of its ingredients to, although originally thinner than, the serum of the blood; a fluid which, apart from the accidental admixture of exsudates, pus-cells, blood-globules, epithelia and the like, contains nothing beyond unorganized effusions of albumen, pigments, fats (cholesterin), and salts. Under no conditions has it of itself alone the significance of a blastema.

It consists, chemically speaking, of water, albumen, fat and extractive matter, and of salts, the chloride of sodium preponderating over the rest, namely, the carbonates and phosphates of alkalies, and of alkaline earths. Generally speaking, its proportion of water is greater than that in blood-serum. The albumen is subject to the greatest fluctuation, down to an infinitesimal allotment.

This relation is liable to various and not unfrequent deviations.

A red coloration is due to blood-pigment.

A yellow, or yellowish-green coloration, to bile-pigment.

A whey-like turbidness, a milky-white appearance, may be owing to certain of the admixtures adverted to, such as epithelium, but especially fat, and to an albumen precipitated by an excess of water [relatively to the saline contents].

Sometimes the fluid has a faint acid reaction.

A notable proportion of albumen renders the fluid viscid, adhesive, synovia-like.

This albumen, for the most part, shows itself to be pure; and not to differ from that of blood-serum; or else it exists as albuminate of soda. It occurs, however, in certain other tolerably well-known, and without doubt in many other as yet unknown, modifications.

Occasionally the dropsical fluid contains urea. The accumulation of the fluid in the textures constitutes *œdema,* of which species of infiltration every organ may become the seat. Its collection in cavities of the body, or of organs, constitutes the *dropsies.* Moreover, the serous effusion developed beneath the vesicated epidermis in erysipelas, in burns, and through the agency of cantharides, is deserving of general mention here.

The mode of origin of dropsy varies:

(*a.*) The *purest dropsy* arises from *retention of the blood in the veins through mechanical hinderance to the circulation.* Its extension varies according to the seat of the obstruction, being considerable in proportion as it affects the centres of the circulation. It is in all probability the veins, even the larger ones, which, in a dilated and thin-walled condition, suffer an out-throwing of dropsical fluid to take place from the blood. The exsudation will be considerable, proportionately to the amount of hydræmia—that is, of the serous crasis—that prevails.

Dropsy is, without doubt, determined by the lymphatics in a similar way.

(*b.*) Nor is there any doubt that serous effusion takes place, in like manner, from the *capillary vessels.* This mode of occurrence applies to the dropsy resulting from general debility; to that arising in palsied parts; to that referable to hydræmia. Again, we may attribute to the same source that acute or chronic œdema consequent upon mechanical capillary hyperæmia, both active and passive; and, lastly, that œdema founded in a slight degree of stasis, or attendant upon consummate inflammation. Of the latter description are those out-pourings of the blood-serum precursory to the genuine exsudation of plasma in the inflammatory process; the œdema encircling areæ of inflammation; the aforesaid serous collections beneath the epidermis, in erysipelas, in burns, &c.

(*c.*) In fine, *dropsical effusions* are brought about by *atte-*

nuation of the blood,—or the serous crasis,—a condition frequently combined with the aforesaid causal influences.

The consequences of serous effusion vary greatly according to the nature of the organs or textures concerned, to the extent of the accumulations, to the acute or chronic form of their occurrence, and to their duration. The *relation of the textures* generally is of much interest. In acute dropsy the textures are in various degrees congested, reddened, and withal—more especially the lung texture—lax, easily torn;—very delicate textures, for example, that of the brain, softened and destroyed. In chronic, enduring dropsy, on the contrary, they are discoloured, pallid, bloated with imbibed serum. Smooth membranes become turbid and dull, the contractile fibre paralysed.

Dropsical fluid is either wholly or partially re-absorbed, or continues unchanged. In the second case, the watery part being first of all absorbed, it becomes concentrated to an albumen, a synovia, or a thin jelly-like mass.

In what manner the various œdemata and dropsies may become perilous, and eventually prove fatal, is sufficiently evident.

B. FOREIGN BODIES.

Inanimate foreign bodies are not unfrequently met with in the organism.

They are introduced accidentally or designedly, either through the natural orifices, as the mouth, the anus, the orifices of the urethra and vagina, the ears, the nostrils; or else by violence, at various parts, as by means of projectiles, of puncture, of a blow, of cautery, &c.

They include things the most dissimilar, as fish- and other bones, fruit stones and seeds, coins, rings, natural and artificial teeth, straw, ears of grain, pencils, needles, tobacco-pipe fragments, dagger and sword points, knives, gunshot materials of all kinds, fragments of dress, of glass, pigments.

These foreign bodies are often got rid of, sooner or later, through the natural channels. Occasionally, however, they abide long—it may be for life—without occasioning serious annoyance, and are afterwards found to have become isolated within a callous exsudate-capsule.

In other cases, they give rise to various, more or less perilous symptoms, and not unfrequently prove fatal. Thus, they may act as plugs, or injure in many other ways every variety of organ. Again, they may induce and sustain inflammation and ulceration to the exhausting point.

A certain interest attaches to the migrations of foreign bodies, as now and then witnessed in the case of needles, grain-ears, and bullets; these being, after a longer or shorter interval, discovered or, perhaps, spontaneously ejected through suppuration, at parts of the body remote from the point of their introduction. These migrations are sometimes the result of gravitation, as in the case of bullets. At other times they are obviously quite independent of this motive power.

c. PARASITES.

Under this generic term we comprehend such formations, infesting the organism both within and without, as represent independent entities, either from the vegetable or animal kingdom. Their investigation belongs to pathological anatomy in general, but especially so, inasmuch as the presence of parasites not only implies previous, but also engenders new, morbid conditions. Moreover, they merit a place in this chapter because it is daily becoming more clear that they are not the production of a *generatio æquivoca* out of diseased organic matter, but that they enter into the organism from without, and find there a soil appropriate for their subsistence and growth.

Parasites are introduced into the organism either as seeds, as ova, or in a more advanced condition,—to germinate, become developed, or grow, in or upon the organism. Nor is it less evident that certain pathological states determine a disposition, not exactly to the generation, but to the evolution and redundant growth of parasites, for which they furnish the necessary conditions. Thus, parasite plants (fungi) readily and commonly germinate in particular exsudates upon mucous membranes, whilst upon normal mucous membranes their sporules remain undeveloped.

Parasites become pernicious in various ways.

I. Parasite plants—Epiphytes, Entophytes. These all belong to the lowest forms of plants, the fungi, and unless collected together in redundant growth, they are too minute to be cognizable with the naked eye.

Respecting their origin by propagation through sprouts and sporules hardly a doubt can exist, and as little as to their translation upon and into the organism; although only in a few instances has it been possible to certify this by direct evidence.

It is obvious that certain conditions are requisite for the harbouring and the evolution of these germs. This often manifestly consists in pathological conditions, and, at the same time, often in processes of decomposition (fermentation, putrefaction). In the great majority of instances, however, we are in the dark concerning those conditions, and the success of our experiments is dependent upon chance. The former contingency is exemplified in fungi upon muco-membranous exsudates, sloughs, and upon mortifying patches of the common integument.

The relation of the vegetable parasite to the concurrent morbid condition varies. The latter sometimes stands in that of a pre-existent state, favorable to the development and multiplication of the fungi; at other times the parasite, harboured through influences unexplained, may become the cause of textural disease; for example, inflammation, suppuration, decadence and loss of hair, &c.

Herewith, the injury they inflict upon the organism terminates. Still they may, where they vegetate extensively, become further mischievous by increasing or specifically modifying some process of decomposition. We may instance the fungi of aphthæ.

If we except the torula cerevisiæ in the contents of the stomach and intestines, the torula of diabetic urine, and perhaps, Goodsir's sarcina ventriculi, (possibly an infusorium[1]), parasitic plants, in man, affect the common integuments and mucous membranes only.

With their buds shooting out into more or fewer long, linked, branched threads, they present the form of the thread-fungus.

1. FUNGI UPON AND WITHIN THE COMMON INTEGUMENT.

The most important are:—

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[1] [It is now however pretty generally admitted to be an alga.—Ed.]

(*a.*) The *mycoderma* in tinea favosa (Schönlein, Gruby). Shut up in splitting capsules, it constitutes the skin-imbedded favus. These fungi, like the torula cerevisiæ, present in their most simple form, roundish or oval cells, and these put forth buds, which shoot out into simple or branched threads. The *favus*-fungus belongs to the genus *Oidium*, (Linck), and according to Müller, greatly resembles the oidium aureum of wood. Or, according to Corda, it may, together with all thread-funguses, which fructify by simple separation of their links, and in which every link may become a spore, be taken, along with yeast funguses into the great genus *Torula*.

There is as yet no certainty as to the part played by these thread-funguses. Attempts at inoculation have hitherto failed, with the exception of one experiment made by Remak.

(*b.*) *Fungi* in the *root-sheath* of the hair in *sycosis* [mentagra, Gruby]. They collect around the hair itself within the root-sheath, and are marked by redundant spore-formation. The spores are spherical, and the thallus-threads frequently contain in their interior little granules.

(*c.*) Fungi in the interior of the hair-roots [Gruby]. In alopecia circumscripta, areata, [porrigo decalvans] the falling out of the hair is caused by a thread-fungus, called by Gruby, on account of the minuteness of its spores, *microsporum*.

(*d.*) In plica Polonica, Günsburg has detected, in the hair-bulbs, a fungus which differs from that of favus.

(*e.*) In Pityriasis versicolor, Eichstedt has discovered a thread-fungus.

Fuchs, Klenke, Helmbrecht have observed a fungus formation in lepra alphoides, and inoculated it with success.

Langenbeck met with a fungus in crusta serpiginosa.

Finally, the mould formations upon sloughing ulcers, and in senile gangrene, come under this head. They are both frequent and copious.

2. FUNGI UPON MUCOUS MEMBRANES.

These are very often found upon the mucous membrane of the mouth, the pharynx, the œsophagus, the intestinal canal, that is to say in fibrino-croupous, and especially in corroding, aphthous exsudates. Aphthæ and diphtheritis of the mouth and throat; croupous exsudates in the same localities, in florid phthisis; croupous exsudates in the larynx, œsophagus, &c. in the sequel to typhus.

They are assuredly not the morbific agent. The croupous exsudates upon which they vegetate are cognizable to the naked eye, for those familiar with the subject, by a peculiar character, consisting in a viscid, curdlike turgescence, dingy yellow or tawny discoloration, and a broken or jagged aspect.

The fungi resemble those of favus. The thallus-threads are, however, mostly much longer, more slender, and have frequently at their free extremity protuberances replete with minute granules (spores). They often form very compact, feltlike tissues.

Amongst these funguses are, no doubt, to be counted the fungus Noma, of Klenke; those found by Bennett in the sputa and lungs, in a case of pneumo-thorax, as also in the black sordes upon the teeth, in typhous patients; again, those seen upon so-called typhus-ulcers in the intestines; lastly, the mould such as we ourselves once observed upon an old blood clot, unattached within a bronchial sac.

II. Parasite Animals (Siebold.)

Parasite animals are divisible, although not strictly so, into ecto-parasites (epizoa), and into ento-parasites (entozoa). The former infest the surface of the body, the latter its different cavities and parenchymata.

Some of them are parasitic during their entire existence; others only at certain periods of it. For this purpose the latter migrate, and enter into various metamorphoses.

Some of them inhabit exclusively certain definite parts of the body, both cavities and parenchymata, others on the contrary occur in various regions of the body, and in great numbers all at once. All this is contingent upon their habitudes, and perhaps still more upon the mode in which they obtain access to their place of abode.

With regard to the difficult question of their origin and propagation, modern researches in the least promising domain, namely, of the helminthes, have pretty well succeeded in subverting the older doctrine concerning the generation of parasites, and their relation to the animals which they infest.

They get introduced into the organism as ova, as larvæ, or even as developed creatures; and wherever they meet with a nidus congenial to their nature, live and thrive upon it. For this habitation to last, however, a peculiar disposition on the part of the subject is no doubt indispensable. In the different parasites this sort of predisposition differs materially. Much is assuredly not required to incur a visitation of ascarides. On the other hand, notwithstanding the extensive dissemination of the ova of the helminthes, the disproportionately small number of persons affected with worms; the circumstance that, under certain conditions (for example, disease), worms for the most part, if not altogether, abandon the individual they had infested; and lastly, the fact that different kinds of worms are proper to different animals; testify to the necessity of the peculiar disposition adverted to, existing in persons affected with worms.

1. INFUSORIA.

The most frequent are the *vibriones*, in purulent and other protein-fluids in the progress of decomposition. Donné has detected a vibrio in the pus of chancre, and rated it beyond its worth.

In pus, the *vorticella*, and also the *colpoda cucullulus* (Vogel) occur.

The *trichomonas vaginalis*, detected by Donné in the vaginal mucus of syphilitic females, is probably not an infusorium, but a misshapen ciliary cell from the uterus or the tubes.

Lastly, we have to cite the *hæmatozoa* occurring in the blood; if they be not rather the embryones of worms, which is probably the case with many of them.

2. INSECTS.

Besides the various *flies* which infest putrid ulcers with their ova and maggots, and the exotic [still problematic] *œstrus hominis*, we have the *flea*, the *lice*, and the *bug*.

(*a.*) *Pulex irritans*, the common flea,

(*b.*) *Pulex penetrans*, the sandflea, common in the West Indies and in South America. The impregnated female burrows into the skin, especially beneath the toe nails, where the brood gives rise to malignant sores.

Of *lice* there are—

(*a.*) *Pediculus capitis*, the head-louse;

(*b.*) *Pediculus pubis*, the crab-louse, infesting, the scalp excepted, every hairy part, and penetrating the skin with its head.

(*c.*) *Pediculus vestimenti*, the clothes-louse, infesting parts of the body devoid of hair, and uncleanly vestments.

(*d.*) *Pediculus tabescentium*, the louse of wasting disease, in which it occurs in great multitudes. The notion, however, that there is a disease in which lice are generated beneath the skin, is without doubt fallacious.

Of *bugs*, we have only to mention the ordinary bed-bug, *cimex lectularius*.

3. ARACHNIDA, ACARINA.

(*a.*) The itch-mite, *acarus scabiei, sarcoptes hominis*, punctiform, from a quarter to half a millemeter long, ovoid, garnished with transverse, bandlike, dorsal striæ, and with central, acuminate warts; anteriorly a bristled proboscis, prolonged inferiorly to a band upon the thorax; four bristly fore-feet terminating in disc-plate, whilst the four hind-feet taper into lengthy bristles.

It burrows in the epidermis, often boring beneath it a canal several lines long, at the termination of which the acarus is, on a narrow inspection, discoverable as a minute whitish speck, marked with a brown point. When the said canals penetrate to the cutis, they engender the itch-vesicles and pustules.

Researches into the natural history of this mite, together with the results of extended experience, prove beyond a doubt its relation to itch as its sole cause.

The follicle mite, *acarus commedonum* sive *folliculorum*, an elongated acarus, from one fifth to one third of a millemetre long, and about one twentieth broad, the head having two lateral antennæ and an intermediate proboscis. The head passes immediately into the anterior part of the body, which occupies about one fourth of the entire mite. From it project four pair of very short, thick, conoid, three-jointed feet, each furnished with three toes. The anterior body passes without break into the posterior, which gradually tapers, but is rounded off at the extremity, is transversely striated, and contains a finely granular, brownish mass.

It inhabits singly or numerously the hair sacs and sebaceous follicles on various parts of the person. Amongst other anomalies, it occasionally displays only six feet, which no doubt implies an earlier state of its development. Its presence is probably often of little moment. Occasionally, however, it may, by stimulating the secretion, engender commedones, or set up inflammation, and thus give rise to the acne pustule.

4. INTESTINAL WORMS. HELMINTHES. ENTOZOA.

Restricting ourselves here to the consideration of such as are peculiar to man, we would preface our special description of them with the following general remarks:

(*a*.) Intestinal worms, *in their consummated development*, are all provided with organs of generation. Those in which the latter have not been demonstrated, are propagated by buds or by offshoots, if they be not imperfect, that is, either larvæ or diseased animals. As opposed to the doctrine of equivocal generation, these facts are important, if we consider:

(*b*.) The migrations and the attendant metamorphoses of the helminthes.

The migrations of the helminthes consist, first in the search for a suitable animal to inhabit, and in introducing themselves into it, when found, through channels formerly unthought of. Secondly, they consist in abandoning the animal dwelt in, for the purpose of casting their ova under favorable conditions, then in passing through one of their metamorphoses, and lastly in searching for another animal for their habitation. They pass, under various phases of development, for the most part through natural orifices of the body, more especially into and out of the intestinal canal. Their occurrence, however, even in the parenchymata, is intelligible upon grounds of direct experience. As illustrative of this, the larvæ of cercarioid trematoda, and of the tetrarhynchi, have been observed to migrate through the parenchymata of mollusca and fishes. It is also deserving of notice, in this place, that helminthes may reach, and settle in any parenchyma through the circulating channels, probably by boring for themselves a passage into the blood-vessels of the intestinal canal. This applies to the nematoid, thread-like animals found by Valentin, Vogt, Gruby, Ecker, and others, in the blood of frogs, dogs, and ravens, and probably representing the embryones of helminthes.

This migration of the helminthes may involve frequent aberrations, and these in their turn many phenomena, which an extended inquiry will perhaps correctly set down to a morbid condition. We refer more particularly to the encysting, the atrophy, and the deformity of certain helminthes; amongst others, of the trichina spiralis, and the tænioid cystica.

(*c.*) The metamorphoses of the helminthes, coincident with their migrations, are of the greatest interest. They constitute a circle of generations, which Steenstrupp, following up the investigations of other naturalists, has pointed out in the trematoda (as in the medusæ, bulb-polypi, and salpæ). A parent animal produces a brood altogether dissimilar to itself, nor identified with it until after three or four generations. These intervening generations of larvæ—these pro-nutrices and nutrices—form without sexual mediation, and are the source of the numerous fallacies taught by the older helminthologists.

(*d.*) All this accords perfectly well with the strict limitation of certain worms to particular countries. The most striking example is afforded in those two riband-worms, the botryocephalus latus, of Russia, Poland, Prussia up to the Vistula, and Switzerland; and the tænia solium of the remainder of Europe.

(*e.*) On the other side, the doctrine of the origin of the helminthes out of intestinal mucus and the like, has not a single point of real evidence in its favour. A disposition to worms exists only in so far as an organism abnormally nourished offers to helminthes, introduced into it from without, a nidus well adapted for their development.

In mankind the following helminthes occur:—

NEMATOIDEA, ROUND WORMS, THREAD WORMS.

Filaria medinensis, the thread or Guinea-worm, of about the thickness of packthread, whitish, from half a foot to several feet long, at the broader end obtunded, terminating behind in a pointed curve. Peculiar to tropics of the Old World, but especially to Guinea; inhabits the subcutaneous areolar tissue, especially of the lower extremities, but occasionally also of the scrotum, the trunk, and the throat. Having spent its earlier period out of the body, it burrows beneath the skin, where it tarries in the areolar tissue for a considerable time, (several months), after which it again perforates the skin from within, in order to disburden itself of its offspring, or in order, it may be, to migrate for this purpose altogether. These proceedings are attended with inflammation and ulceration, and great caution is recommended, in any attempt to extract the worm, to avoid tearing it; either the elapsing brood or other contents of the worm, having an erosive property which tends to aggravate the said processes. It would seem that, as yet, none but females have been observed. Accordingly these must have introduced themselves in the impregnated state.

Trichocephalus dispar, the hairhead or whip-worm; filiform; the anterior part hair-like, the posterior part considerably thicker; from one and a-half to two inches long; of distinct sexes. The male is, at its posterior part, spirally convoluted, and its penis contained in an elongated, funnel-shaped, violet-coloured sheath. The posterior part of the female is not spiral.

It infests the cæcum, singly, and also frequently in multitudes [especially, it is true, in the dead bodies of persons who have died of protracted typhus or similar diseases], without occasioning any extraordinary symptoms. The females are loaded with ova, which are, however, not developed in this locality.

Ascaris lumbricoides, the cylinder worm, a widely disseminated intestinal worm, from five to twelve inches in length, cylinder-shaped, tapering towards both extremities, especially towards the anterior; having four longitudinal striæ, two of which are more strongly pronounced; densely marked with transverse striæ; semi-diaphanous, so that the intestinal canal and the organs of reproduction are transpicuous. The head, divided from the body by an annular groove, displays three little nodules, or rather valves, which encircle the mouth. The caudal extremity, especially in the male, is incurvate. Sexes distinct, the male being smaller and narrower, and having at the caudal extremity a thin, capillary, sometimes double penis. The female is larger, and exhibits at its upper third a fissure from six to eight millemeters long, as the orifice to the organs of generation, which contain ovaries and oviducts of enormous length.

It infests the ileum, often in extraordinary number, in groups and conglomerate masses. A brood is never met with; the ova, therefore, become hatched extraneously to the human body, to remigrate thither afterwards, as the living brood. It gives rise to the well-known worm symptoms. The perforation of the intestine, however, [migration extraneous to the intestinal membranes] and its sequelæ are, to say the least, extremely rare.

Oxyuris vermicularis, (the *Ascaris vermicularis* of Rudolphi) the hook-tail, maw-worm;—a little, thin, white worm. Sexes distinct. The male very rare and small, from one to one and a-half millemeters long, with spiral convoluted tail; annulate; with a tail terminating in a fine transparent point. The head of either displays a transparent swelling, which under the microscope appears as a wing-like membrane.

It inhabits the colon and especially the rectum, occasioning both here and in the vagina, into which it creeps, an intolerable itching. As it is never accompanied by a brood, it probably migrates as the impregnated female.

Strongylus gigas, [Pallisadenwurm.] Giant strongle; a very large, cylindrical worm, of from five inches to three feet long, and from two to six lines in thickness; when recent, of a fine red colour. Sexes distinct; the male smaller, more tapering towards both ends; annulate, with shallow, longitudinal grooves; head obtuse, with six papillæ; at the tail extremity, a funnel-shaped pouch, out of which protrudes a very thin penis. The female, larger, with obtused caudal extremity, and near it the vulva.

Inhabits the kidneys; is rare both in man and in brutes; [found in the dog, the wolf, the marten, the horse, &c.]

To these may be added the following nematoda and nematoid pseudo-parasites, some of them being very rare, or even but once met with.

The *filaria bronchialis*. [*Hamularia lymphatica*, Treutler—*H. sub-compressa B.*, once seen by Treutler in a degenerated bronchial gland in the human subject.] *Filaria oculi humani*, [in the liquor Morgagni and in the cataractous lens, Gescheidt, Nordmann.] The *filaria* in the blood, [Klenke]; the *anchylostoma duodenale,* [Dubini, in the duodenum]; the *spiroptera hominis,* [Barnett, in the urine]; the *dactylius aculeatus,* [Curling, in the urine]. Finally the *encysted nematoda.*

Trichina spiralis, an incarcerated worm, which one might be tempted to class intermediately between the nematoda and the cystica, were it not extremely probable that it is only a strayed nematodon which, without coming to maturity, encysts itself, perishes, and cretifies within a second cyst thrown out from the textures.

The worm is enclosed within a double cyst, an external one, mostly lemon-shaped, and an inner, oval one; the space of the first, at its two ends, being filled up with very fine, dark granules. Both consist of a homogeneous, faintly granular structure; the former being about one fiftieth of an inch long, and one ninety-fifth broad, the latter one seventy-seventh long. In the inner cyst, amidst a more or less granular, viscid, transparent fluid, lies the worm, perfectly free, and generally rolled up in two and a half spiral convolutions. When extended it is from one twenty-fifth to one thirtieth of an inch long, and about one six-hundredth broad, lumbricoid, thread-like at both extremities, although more pointed at the one than at the other. It possesses internally a winding canal, interpreted as intestine, and a granular organ, the designation of which, as an ovary, is, without doubt, erroneous.

Occasionally the cyst contains two, or even three, worms.

The *Trichina spiralis* inhabits the voluntary [striated] muscles, and always in vast multitudes, the muscles appearing to the naked eye studded with little white specks. The cysts always lie with their long diameter parallel to the course of the muscles. [Hilton, Owen, Blizzard, Henle, and others.]

TREMATODA, SUCTION-WORMS.

Epecially characterised by their peregrinations and metamorphoses.

Distoma hepaticum, and ***D. lanceolatum,*** Liver-fluke; flat, melon-seed or lancet-shaped, soft worms, of a yellowish white colour, with two suction pores; one of which is seated at the head extremity; the other, which terminates cæcally, at the belly. Between the two is the sexual orifice. They are hermaphrodites.

The *Distoma hepaticum* is the larger, being from four to eight or to fourteen lines long, and from one and a half to six broad, with a branched intestinal canal.

The *Distoma lanceolatum*, as the smaller, is from two to four lines long and about one broad. Its intestinal canal is bifurcated.

Both infest the liver of the herbivora, rarely of man. The *D. lanceolatum* has only once been met with in the latter. In brutes they occur in great multitudes, obstructing and dilating the gall-ducts.

Distoma oculi humani. A minute distoma, once met with in a child between the cataractous lens and its capsule.

Polystoma pinguicola, Hexathyridium pinguicola, (Treutler). An inch long and from two to three lines thick, oval, superiorly convex, inferiorly depressed worm, with six pores at its head extremity, and a larger abdominal aperture anterior to the tail. Found once by Treutler in the fat of an ovarian fat-cyst.

Polystoma venarum, Hexathyridium venarum (Treutler), probably a pseudo-parasite.

CESTOIDEA—TAPEWORMS.

These are characterised by their enduring growth, and by the great length to which they attain. They consist of a succession of linked joints, of which the fully developed, sexually mature, hindmost ones become cast off in greater or lesser series; whilst at the neck, fresh joints are continually being reproduced. As in these, again, a brood is rarely seen associated with the old individuals, whilst the separated, sexually mature joints so frequently become ejected, it is probable that the embryones become developed externally to the animal they infest, to re-immigrate subsequently.

In mankind there occur:

The *Tænia solium, T. vulgaris, T. cucurbitina*, the ordinary tape-worm, long-jointed tape-worm, chain-worm; a white, or yellowish-white worm, twenty feet long and beyond it, anteriorly thin, roundish,—posteriorly flat, and from three to six lines broad,—jointed. The joints are flat, square, towards the distal end more and more oblong-square, resembling gourd-seeds with truncated apices. At the right or left margin, often alternately, is seen a wartlike projection marked by a pore with a raised brink. This is the orifice of the sexual organ, which represents a cavity dendritically branched throughout the joint. The head constitutes at the very thin anterior termination, a nodule-like intumescence, with four lateral, black points in relief. There are four suction pores; and, between them, is seated upon a slightly raised circle a double coronet of hooklets. The annulate neck is studded with numerous lime-corpuscles of the most various size. (vide *Cystica.*)

Inhabits the small intestine in man, almost in all districts, except where the botryo-cephalus occurs. The belief that it only occurs singly in man is quite adverse to experience. We have discovered nine of them in the corpse of a lad. It occasions the well-known annoyances, but no visible anatomical mischief.

Botryo-cephalus latus, *tænia lata*, the broad or broad-jointed tape-worm, resembles the last in many points, equalling it in length, and being in like manner jointed. Its joints are usually broader than those of the *T. solium*; this alone, however, cannot pass for a diagnostic mark. The wartlike projections are not, as in the other worm, seated at the margin, but at the centre of the ventral surface. Their pore leads to a branched, rosette-shaped, sexual organ. The head, differing from that of the *T. solium*, exhibits no suction-pores, but two longish grooves.

Inhabits the small intestine in man, but is strictly limited to Russia, Poland, Prussia [trans Vistulam], Switzerland, and the South of France. If it occur elsewhere it is assuredly imported from one of those countries.

It rarely parts with single joints or links, but usually with a greater or lesser chain of them.

CYSTICA. VESICULAR WORMS.

In the formation of their head, these resemble tapeworms to such a degree, that even in 1836 Johannes Müller proposed to unite them in a single order, with two subdivisions. Light has, however, been since thrown upon the subject, which warrants us in going a step further, pronouncing these cystica with tape-worm heads to be in truth nothing more than *errant cestoda*, which, owing to their deviations, have sickened, declined and remained, sexless.

The lime corpuscles found upon them, and especially upon the cysticercus, are the same as those occurring upon tape-worms. They have been erroneously held to be ova, and in reality rather represent an outer skeleton formation. These cystica, within textures, are almost always distinctly encysted; that is, shut up within a capsule effused from the textures. In free spaces,—for example in the ventricles of the brain, this is not the case. This adventitious outer cyst is not to be confounded with the cyst proper to the animal itself. They frequently perish, especially through inflammation of the external cyst, being either mechanically crushed by, or corroded and destroyed in, the product. In the sequel, the complicated contents of the outer cyst, after having suffered many changes, progressively thicken, and eventually cretify, *en masse*, within the shrivelled capsule.

The unequivocal proof of the previous existence of an animal in such obliterated cysts is furnished by débris of the animal cyst; by hooklets, from the coronet of hooklets, which have resisted the corrosive agency; and lastly, by the presence of the lime corpuscles before alluded to.

In man occur:

The *cysticercus cellulosus*, consisting of a conical, snow-white, transversely rugous body, and of a vesicle which constitutes its caudal extremity. The vesicle is oval, spherical or square,—in muscles, cylindrical, parallel to the muscular fibres,—and of the size of a pea or a haricot bean,—in rare instances, for example, in the ventricles of the brain, of a hazel nut. When the animal is retracted into this vesicle, it appears as a white, spherical, solid body, seated somewhat eccentrically on its inner surface, whilst upon the vesicle itself is observable, externally, a delicate pointlike fold or depression at the same spot. When the animal is external to the vesicle, a condition easily brought about by puncturing the vesicle, and pressing the hardish spherical body between the finger and thumb, a pore becomes perceptible which leads to the interior of the animal pouch. Taking the size of the caudal vesicle at the ordinary one of a pea, the animal itself, that is the trunk, would about equal the diameter of the vesicle, both together measuring from six to twelve lines in length. The neck is short, very thin, and, like the body, wrinkled. Upon it is seated the largish, bulb-shaped, or rhomboidal head, upon which there is at each angle a circular suction-cup; and midway between these a proboscis, cone-shaped in its protruded state, with, at its extremity, a coronet of hooklets consisting of a double row [about thirty-two in all], which, when retracted, pack up into a funnel-shaped cup. The two circles of hooklets are identical in shape; those of the outer circle are however much smaller than the others, whilst both are so disposed that the larger and smaller hooklets alternate with each other.

The above-mentioned transversely wrinkled, anterior portion of the creature appears as an almost structureless, feebly striated membrane, to which a profusion of fine, black-contoured molecule adheres. It is, moreover, studded with a multitude of roundish or oval, whitish, smooth, sharply contoured, shining, lesser or bigger corpuscles, of from one eightieth to one thirtieth of a millemeter in diameter. They are most numerous about the middle part; near the neck and head their number greatly diminishes, whilst, close to the caudal vesicle, they suddenly and entirely disappear. They lie superimposed in several layers, those of the outer stratum being only loosely adherent to the animal, so that they may be very easily scraped away. Treated with hydrochloric or with acetic acid, they dissolve under the copious development of carbonic acid, leaving an organic base-substance behind. In the solution, oxalic and sulphuric acids create a precipitate.

The caudal vesicle consists of the same homogeneous, indeterminate, granulated mass, besprinkled with countless small and larger fat-molecules. The contents of the caudal vesicle consist of a watery, neutral fluid, holding but a scanty portion of albumen.

Wherever the cysticercus occurs in textures, it is inclosed within a second cyst of fibrous texture. When magnified it appears as a delicately-fibred membrane, permeated by delicate blood-vessels, and easily rendered transparent by acetic acid. Where the cysticercus occurs free within a cavity, as within the ventricles of the brain, it is uninvested, showing the outer cyst, in other localities, to be adventitious.

When the creature perishes, as frequently happens from disease of the outer cyst, the caudal vesicle becomes semi-opaque, collapsed, its contents turbid, displaying the said lime-corpuscles and hooklets, which, together with a granulate mass, are found floating in its fluid. The entire creature softens and liquefies, afterwards condenses, and eventually settles into a cretaceous concrement. Meanwhile the outer cyst shrivels and dwindles into a thick-membraned capsule, for the isolation of the said concrement.

The cysticercus cellulosus occurs in the brain, in the striated muscles, including the heart, and in the areolar tissue. It also occurs, free, without its outer envelope, in the ventricles of the brain, and in the chambers of the eye. It sometimes occurs in the muscles and brain simultaneously, in great multitudes.

Even in the brain it is usually borne imperceptibly. When present there in great numbers, however, it often occasions vertigo, and the case has happened of its proving fatal by setting up inflammation in its vicinity.

Echinococcus hominis and *acephalocystis* (Laennec). The relation of both to each other, and the import of the last-named animal in particular, will become manifest from the following *description:*

(*a.*) *Echinococcus.*—Within a sac of fibroid texture is inclosed a solitary, independent, thoroughly distended vesicle, containing a limpid, serous fluid; or else inclosing, as a parent vesicle, other similar vesicles of various size, in various numbers, spherical or flattened by mutual compression, either floating at large in the contained fluid, or sessile upon the inner membrane of the said parent. Its size varies from that of a vesicle just cognizable, and as big as a poppy- or a millet-seed, to the magnitude of a goose's egg and more. In number it may amount to hundreds, so that the serous contents of the parent vesicle are reduced to a minimum. Generally speaking, the lesser filial vesicles are sessile, whilst the larger ones are free.

In very voluminous sacs it is common to find that the parent vesicle appears to be wanting. Either it is mixed up with the younger vesicles, split up, collapsed and dissolved into scattered shreds, or else it has disappeared in the excessive attenuation consequent upon its enlargement.

In their unimpaired vegetation, these vesicles are filled to distension, are elastic, and impart to the touch a sense of tremulous fluctuation, as does the parent cyst replete with them [hydatid tremulousness]. They consist of a substance resembling coagulate albumen, separating into several layers, partly diaphanous, partly white and opaque, frequently accumulated in the inside to considerable thickness, and into gibbous projections. Moreover, they contain a limpid serosity identical with the contents of the parent cyst. When the vesicle is punctured, this fluid gushes forth in a column, and on an incision being made, the parietes of the vesicle become suddenly inverted. The substance of the latter is a stratified, homogeneous, very fine-granular, structureless mass, whilst their contents exhibit a few lustrous fat drops, some, scattered or agglomerate, elementary granules, and glebous coagula.

These vesicles occasionally contain others similar, of a third, and the latter again in rare instances of a fourth generation.

On a narrower inspection of the inner surface of these vesicles, we perceive in many of them, a whitish, opaque, gritty efflorescence, whilst with the aid of the microscope we here discover densely nestled animalcules, which prove, by the most various changes of shape, that they long continue to live on in the dead subject. A few of them are even found free in the above-mentioned fluid.

This entozoon is from one ninth to one third of a millemeter long, and from one twelfth to one fourth of a millemeter broad. It has a tænioid head, with four lateral suction-pores, and a proboscis garnished with a double coronet of hooklets. The head is distinguished from the thicker, spheroid trunk, by an annulate indentation. From the proboscis a longitudinal striation runs to the posterior part, and, commencing from these striæ, the body of the creature is transversely striated. The posterior termination is a transverse cleft, in which is inserted a cordlike formation, by whose means the creature maintains its seat upon the vesicle. Between the striæ of the trunk are spherical or oval, limelike corpuscles, resembling those upon the cysticercus.

In its developed state the creature appears in the above form. It is met with, however, under various other shapes.

Thus it appears as an elongated sphere, in the centre of which the coronet of hooklets appears perspicuous when the head is retracted. Or it assumes the shape of a heart, or of a pitcher, or even of a horse-shoe.

The abode of this echinococcus in mankind is, according to our own experience, invariably internal to, and never external to, the vesicles.

(*b.*) *Acephalo-cyst.* Under this term we at this day understand nothing beyond those vesicles which we have just described as being inhabited by the echinococcus, but which are in some instances sterile. The above name has been given to this formation in order to designate that supposed independent vitality which the absence of organs still renders problematical. The acephalo-cyst, which Blainville reckons amongst the "monadaires," and Kuhn compares to Agardh's protococcus, with its multiplication by buds, is in our own opinion not to be held separate from the echinococcus, although the precise relation between the animal and the vesicle is by no means clear.

The relation of the primary acephalocyst [the echinococcus-vesicle] to the outer cyst, is analogous to that of a new-growth incapsuled by exsudation from the surrounding textures.

1. W. Griffith has examined acephalocysts and their contents. The transparent fluid, of 1·008 sp. grav., coagulated readily by heat or nitric acid, and contained an inconsiderable amount of fat. A thousand parts yielded fifteen parts of solid ingredients, principally common salt. They left 0·85 per cent. of this salt, a little sulphate of soda, a trace of phosphate of lime, and some albuminous extractive matter, but neither cholesterin nor alkaline phosphates. The envelopes of the hydatids left, when dried, a brown residuum, which dissolved with a deep brown colour when boiled with hydrochloric acid, but was not again precipitated on the addition of an alkali. When moist, they dissolved in hydrochloric and in nitric acid, but the solutions were precipitated neither by ferro-cyanide of potassium, nor by tincture of galls. Nothing was dissolved by boiling them in water, for neither by tannic nor by nitric acid was either any precipitate formed, or the fluid gelatinized. When boiled with carbonate of potash, the dried membranes

were dissolved with brown coloration, but without any accompanying development of sulphuretted hydrogen, nor any precipitation on the addition of an acid.

Acephalocysts, together with the creatures that infest them, are extremely liable to destruction, through hypertrophy, atrophy, and consequent perforation of their external coat; but most particularly through inflammation of the latter with its products.

It is not a rare thing to find, within a sac, individual vesicles imperfectly filled, or collapsed, with walls transparent, tumefied, gelatinized, or even degraded to a smeary mass. The contents of such vesicles are turbid. They consist partly of fat globules with a fine pulverulent point molecule in great abundance, and the débris of broken up echinococci. Occasionally this conversion affects most, if not all, of the vesicles. They burst, or rather open out, owing to the increasing tendency to dissolution, until at length the entire contents of the parent cyst are rendered turbid.

The inflammation of the outer sac, a frequent occurrence, is important. It bears the character of inflammation of a sero-fibrous membrane, and throws its products, for the major part, upon the inner surface and into the cavity of the cyst.

It is in many instances to be regarded as a fortunate event, leading as it does to the disruption and extinction of the acephalocyst, with its inhabitant animalcules, and in due time to the shrivelling and decay of the entire sac. The contact of the acephalocysts with exsudate, and the reception of the latter through endosmosis into the walls and cavity of the acephalocysts appear to be amongst the most ordinary causes of their dissolution. After the effusion, gradual resorption of a portion of the contents—that is, of the original serous fluid, and of the exsudate—follows, whilst another portion thickens to a grayish, unctuous chalk-pap, and eventually cretifies altogether. The sac shrivelling commensurately with the diminution of its contents, becomes obliterated in such wise as ultimately to inclose a mass consisting of variously superimposed residua of acephalocysts (echinococcus-vesicles) and of the said chalk-pap or concrement.

It is not unfrequent for an intense inflammation to terminate in ulceration of the sac, so that an abscess, inclosed within the implicated parenchyma, takes its place. This, together with consecutive suppuration in neighbouring textures, may lead to the opening of the sac into another adjoining one; or to its opening externally; or into one or other of the great serous sacs; into the intestinal canal; into the urinary cavities or passages; the gall-ducts, &c. The direction in which such an abscess empties itself decides the question as to the favorable or the unfavorable issue of the case.

The echinococcus and acephalocyst are particularly frequent in the liver, less and less so in the subperitoneal, areolar tissue, and in the peritoneum, in the omentum, in the striated muscles, including the heart, in the brain, in the spleen [mostly in concurrence with others in the liver], in the kidneys; very rare in the lungs and bones.

Not unfrequently they occur in several organs simultaneously. Thus they will infest in vast numbers both the peritoneum and the abdominal viscera. In magnitude the sacs may attain, or even exceed the diameter of a foot.

The echinococcus-cysts may become perilous through their volume; and, when present in great numbers, prove fatal through exhaustion and general wasting, as also through the aforesaid inflammatory and suppurative processes.

SPURIOUS PARASITES.

As such are to be reckoned all those foreign bodies reputedly or really, accidentally or designedly, conveyed upon or into the human body; but which are proved either not to infest it in reality, or to be of a nature even manifestly to preclude a parasite existence.

We have to bring into this account not alone animal creatures, and various parts of animals and of plants; but also misshapen, diseased textural parts of the organism, or products of disease. Such are:

1. Animals and parts of animals dead or alive, really voided by stool or rejected by vomiting, such as the larvæ of flies received into the stomach with food in a state of decomposition, or accidentally or designedly added to the matter so evacuated.

2. A great variety of other bodies of the descriptions adverted to.

Amongst the spurious parasites of the present day we may cite—

(*a.*) The *trichomonas vaginalis* of Donné,—probably a misshapen ciliary cell.

(*b.*) *Diceras rude* (Rudolphi), repeatedly recognized as the undigested seeds of mulberries.

BLOOD DISEASES—DYSCRASES.

Humoral pathology is simply a requirement of common practical sense; and it has always held a place in medical science, although the limits of its domain have, no doubt, been variously circumscribed or interpreted at different times. Of late years it has met with a new basis and support in morbid anatomy, which, in the inadequacy of its discoveries in the solids to account for disease and death, has been compelled to seek for an extension of its boundary through a direct examination of the blood itself.

Not alone has pathological anatomy demonstrated the existence of blood diseases in unlooked for detail; it has at the same time solved a problem of the weightiest import. It has, we think, decided in favour of a humoral pathology, by demonstrating a primitive anomaly of blastemata; by demonstrating the endogenous impairment of the blood within the vascular system, in the inflammatory process, as the basis of the variations in exudates [blastemata]; lastly, by demonstrating the dependence of local morbid action upon preexistent impairment of the general circulation. Our attention will be here directed to diseases of the blood in its totality, and to local dyscrasial processes, with inflammation at their head, only in so far as these offer the basis and starting-point for consecutive disease of the entire blood-mass. It is remarkable, however, and no less important for practice than for science, that the essential forms of these local dyscrasial processes,—perhaps of all local dyscrasial disease,—occur, likewise, as primitive affections of the entire blood-mass. This is proved by the varied character of the products of the inflammatory dyscrasial process, and a comparison in detail of these products with anomalies of the general blood-crasis. Thus, primitive pyæmia, fibrin-crases, sepsis of the blood, severally occur independently of all local beginning, and of all infection.

There are, indeed, two ways of investigating and recognizing blood-diseases: first, the *anatomical examination* of the blood in the dead subject, or of blood obtained during life through spontaneous or artificial hemorrhage; and secondly, *chemical analysis*. Both kinds of investigation should be supported, and the results controlled, by a concurrent examination of the secretions and excretions, of the general condition of the solids, and of new-formations, especially of such exsudates as are the offspring of inflammation.

In fine, both kinds of research should go hand in hand. For, although a deeper insight into the changes suffered by the blood may be reserved for chemistry, it must needs be based upon sound anatomico-humoral premises. Up to the present day chemistry has not taken this duly into consideration, so that as yet this science cannot be said to have far excelled the achievements of a circumspect anatomical survey, notwithstanding the limited resources at the disposal of the latter.

Upon the chemical pathologist we would strongly urge an unremitting prosecution of his researches. We would recommend him to direct his labours more particularly towards ascertaining the precise character of the impairment suffered by the proximate ingredients of the blood, and of the anomalies impressed upon its elementary composition. The interests of hæmato-pathology would after all, perhaps, be best served by the examination, in the above sense, of blood taken from the dead subject, the diagnosis of the case having previously received the light of a general post-mortem examination.

Our own task in these pages will be limited to establishing a purely anatomical pathology of the blood; we shall therefore restrict ourselves, as nearly as possible, to anatomical facts, although without neglecting to avail ourselves of the collateral support of such chemical data as may be relied upon at the present hour.

It is the business of pathological anatomy to determine both the physical properties of the blood in its totality, and also the relative quantity, and more especially the quality, of its more immediate components. The two main components which come peculiarly within its province are, first, those essential form-elements, the *blood-globules*; and secondly, the spontaneously separating, coagulating, solidifying *fibrin*,—that component which, owing to its varying tendency to become organized, is, in an anatomical sense, the most important of all. We will here *summarily refer to what has been said in the introduction to the doctrine of blastemata and to the section on exsudates*, and then proceed to treat of *blood diseases* in what would appear to be their most natural order. The subject is, however, so intimately allied to that treated of in the chapters referred to, that a certain amount of repetition will, perhaps, be unavoidable in the following pages.

Affections of the blood are, like those of the solids, either *primitive* or *consecutive*. And again, the former, equally with the latter, suggest an inquiry as to whether they result from an immediate influence of the morbific agent upon the blood, or are determined by the nervous system, as the actual percipient, alienated both in matter and in function. This question can, however, hardly affect us in this place, since the latter view mainly rests on speculative grounds, and upon the fact that obvious injury to, or sensible anatomical disturbance of, the nervous system sooner or later results in disease of the blood.

The latter are determined in very different ways by anomalies in the solids. Thus, the hindered eliminating activity of an organ occasions retention of effete matter in the blood; an abnormal plastic process influences the blood crasis, directly or indirectly, through the anomaly in the interchange of matter. Take for example, the infection of the blood within the range of an inflammation.

To diseases of the solids, as local morbid processes in the broadest sense, affections of the blood stand in a two-fold relation:

1. *The anomalous crasis is a pre-existent one—the primitive affection; the local disease a localization thereof—the secondary affection.* The point of localization, apart from the effect of concurrent external influences, is determined by a specific relation of the crasis to certain organs presided over by the nerves. The forms it assumes are chiefly those of hyperæmia and stasis—inflammation, absolute stasis,—exsudation, or, without the latter, a product-formation completed within the blood-vessels;

for instance, spontaneous coagulation of diseased fibrin, pus formation within a greater blood-vessel or within the capillaries of an organ.

The relation of the various crases to the organs and textures, nay, even to particular sections of organs, is manifold. Thus, the croupous fibrin-crases evince a very marked preference for the mucous membrane of the air-passages, and for the lungs themselves; the typhus-crasis, for the mucous membrane of the ileum; the exanthematous crases, for the common integument and for mucous membranes.

2. The anomaly of the general crasis is consecutive; that is, the consequence of a local disease, and especially of local dyscrasial processes, whereof the products are taken up into and affect the general blood-mass. This happens—

(*a*) Through resorption of the effused products by means of the lymphatics, or immediately into the veins.

(*b*) Through reception into patent blood-vessels. This process includes the reception of products thrown out into the cavity of larger blood-vessels,—pus, for example.

(*c*) Most of all, through the off-flowing, and the return into the veins, of plasma degraded in the local process, in a manner corresponding with the quality of the exsudate. [See "relation of the inflammatory process to the crasis"].

It is, however, to be understood that, neither does a dyscrasis necessarily always become localized, nor a local dyscrasial process invariably give rise to a consecutive dyscrasis of the entire circulation. In the former case, a certain degree of intensity of the dyscrasis is no doubt requisite; in the latter case, the reception of a sufficient quantity of plasma, degraded in the manner aforesaid by the local process, or else of a heterogenously diseased,—for example, of an ichorous or septically constituted—plasma is indispensable.

Blood diseases are, moreover, either protopathic, whereby we mean developed out of the normal crasis, or deuteropathic, that is created out of another anomalous crasis. [Meta-schematism.] Deuteropathic crases occur in the simplest manner, as impoverishment of the blood in one or more ingredients, drained away by excessive deposition into textures or upon membranous expansions.

Blood diseases are both acute and chronic, and they are marked accordingly by the rapidity or by the slowness of their career. This is contingent upon the character of the dyscrasis, upon its grade, and upon the significance of the organ in which it becomes localized.

They issue:

(*a.*) *In transition to the normal blood-crasis.* This occurs under various conditions, for the most part obscure; for example, under the return of the free function of an organ, under exhausting localization of the dyscrasis in one of the aforesaid processes, or in some secretion. In this way tuberculosis and cancer may lose their *general* import and become local affections, which either go on vegetating under the normal condition of the blood, or enter into a retrograde metamorphosis.

(*b.*) *In transition to another anomalous crasis,* [meta-schematism.] Such transitions are multiform, some of them appearing to be necessary conversions when the original crasis is at its acme, others to represent the final wearing out of some component of the blood. With respect to others, however, we are still in every way completely in the dark.

(*c.*) *In death,* not only through overwhelming localization in vital organs, often coupled with palsy of their function; not only through exhaustion of organic matter and of the powers of life, owing to redundant local production; but also, in many instances, through unfitness of the dyscrasial blood for the upholding of processes essential to life, for the maintenance of nutrition generally, but especially of respiration and of the energies of the entire nervous system, both central and peripheral.

FIBRIN-CRASES.

The fibrin-crasis occurs in several most important forms and varieties, which the term hyperinosis — as designating a frequent but by no means necessary and invariable excess in the quantity of fibrin—does not sufficiently characterise. It overlooks the far more important, and, for the most part, very marked feature of *quality.* However certain may be the *excess* of fibrin, its qualitative deviation becomes more and more distinctly pronounced in proportion as in the series of fibrin-crases the forms recede from the characters of *true* fibrin. This, with the exception of a few hints thrown out by Andral, has been hitherto ignored. In this qualitative anomaly, however, the varieties of the fibrin-crases are founded. Each may be, and very often is, a hyperinosis, at the same time. Still the qualitative anomaly is the essential point; and it may, in a *hypinotic* crasis—that is, in poverty of fibrin—cling to a minimum of fibrin, and with it manifest at once that peculiar tendency of fibrin-crases to localization, and a marked peculiarity in the product.

These different fibrin-crases are, as in the sequel their special delineation will show, manifested by certain anomalies of appearance and structure. They are distinguished in common by the proneness of their fibrin to coagulate, and by its deposition, more or less pure, within the vascular system, from the heart to the capillaries downwards. They are, moreover, marked by their localization in inflammations which are wont to affect very vascular organs, such as the lungs, mucous and serous membranes, and areolar tissue.

The fibrin-crases including pyæmia, tend more particularly to prove that all those changes which the plasma undergoes in local dyscrasial inflammation, and its products or exsudates, take place within the general circulation and by virtue of its own intrinsic relations, not through any local reciprocation between the blood and the textures inflamed.

The fibrin-crases constitute, generally, the so-called *phlogistic blood-admixture,* against which, conformably with the view of a quantitative exaltation of the vital process, the lancet has ever been opposed. Yet amongst the processes in question, far-sighted pathologists have always discriminated some in which much bleeding appeared not only needless but even mischievous; we refer more particularly to the croupous processes. That in these, a qualitative deviation in the constitution of the fibrin plays the part chiefly deserving of attention, is proved not only by the more obvious anomalies of aspect and of structure before alluded to, but also by—

1. The proneness of these crases to become localized, even where the amount of anomalously constituted fibrin in the blood is very inconsiderable; as, for instance, in the secondary croupous crases emerging out of the typhus-crasis; in the tuberculous crasis, in which the last particle of fibrin is expended in the deposition (localization) of tubercle.

2. The reaction of many congenerous exsudates upon their parent textures,—this reaction consisting in softening and corrosion.

3. The wasting attendant upon hyperinosis, or the predominance of fibrin, that element of the blood commonly held in an especial manner to preside over general nutrition. Here the alienation of functional activity can only be interpreted as qualitative.

We have already adverted to the localization of the fibrin-crases. In relation to this, it is a question equally interesting and opportune, *whether the crases regarded as hyperinoses, in inflammations, be determined by the latter, or constitute the primary and fundamental disease?* The view received in France, tends to demonstrate the dependence of the crasis upon the local inflammation; in other words, the symptomatic character of the crasis. With the setting in of the inflammation and its increase, the amount of the fibrin is supposed to become augmented.

We are fully convinced that an inflammation obviously called forth by external causes may, by the abduction of endogenous, and the resorption of exsuded products, give rise to a corresponding crasis, which will become augmented in proportion as the inflammation increases in intensity and extent. On the other hand, we believe that spontaneous stases are localizations of a crasis, and stand to it in a dependent—a conditional—relation. This opinion is based upon the following facts:

1. Every crasis [by no means the fibrinous crases alone, to which, as the so-called phlogistic, we might be disposed to concede this prerogative] is capable of localizing itself in the shape of inflammation; take for example, the typhous and the exanthematous crases. The objection that inflammations, and especially pneumoniæ, arise during the progress of typhus, is met by the fact that those inflammations with the character of a fibrin-crasis are based upon a fibrino-croupous crasis—in other words, that they are the localization of a fibrino-croupous crasis into which the typhus has become converted. Genuine typhous pneumonia (pneumo-typhus) does not develop a fibrin-crasis, any more than does typhous inflammation of the intestinal follicles or of the mesenteric glands.

An objection of considerable weight against the opinion promulgated by Andral, is furnished by pneumonia, the very process commonly concurrent with the most marked hyperinosis. We believe that ordinary pneumonia [with fibrinous product] is, for the most part, the localization of a pre-existent, that is, precursorily developed crasis, a crasis characterised by an incontestable relation to the lungs, and to the mucous membrane of the air-passages. Such a view does away with the paradox that inflammation of the lungs, a disease which, in its intense form, attacks and disables large sections of the lung, should uphold so enormous a development of fibrin, whilst other lung diseases lead to crases of the very opposite kind—in a word, to venosity [Hypinosis, Albuminosis, Cyanosis.]

3. Lastly, the argument derives force from the general disturbance which always precedes a localization, seeming to bear witness to an alteration in the crasis. And to this may be added, the nature of the causal influences, which appear to be rather general than local. We may instance epidemics, climate, weather, &c.

Fibrin-crases become, for the most part, primitively developed under the conditions of a free respiratory function. Of this fact, striking examples offer in the tuberculoses, and the setting in of croupous and of tuberculous processes after childbirth, that is to say, after the release of the thoracic spaces and of the lungs, resulting from deliverance of the womb.

Certain fibrin-crases are *primary,* and distinguished by their localization upon the mucous membrane of the air-passages, [Laryngeal, tracheal, bronchial, pulmonary croup] upon serous and synovial membranes, and in large accumulations of areolar tissue. Others are *secondary,* that is, the consequence or the conversion of other crases, for example, of the typhus-, of the exanthema-, of the cholera-crasis. In these, a *qualitative* anomaly of the fibrin predominates, as shown by this, that, even where but an inconsiderable amount of fibrin becomes deve-veloped, localization takes place, and this of an unwonted kind, as, for instance, upon the mucous membrane of the intestinal tract, of the urinary passages, of the gall-ducts, &c. Others, again, are *primitive, spontaneous*, or even *consecutive affections* of the blood determined by infection with analogous substances. They are often epidemical.

Above all other crases, the fibrin-crases, like the fibrin-exsudates, are never thoroughly pure. Every portion of morbid fibrin has, associated with it, another portion of less diseased, or even of normal fibrin.

The products of the localized fibrin-crases [endogenous coagulations, and especially exsudates engendered by inflammatory stasis] are partly organizable [designed for regeneration, or expended in hypertrophy], partly unorganizable, liquefying, corrosive, purulent, or ichorous. These exsudates correspond so completely with the nature of the coexistent fibrin-coagula within the vascular system, that the character of the one may with safety be inferred from an acquaintance with the other.

It is interesting that coagulations in the left heart, that is out of arterial blood, are not alone more decidedly compact, but also more frequent than those out of venous blood. As evidence of this we may cite the incomparably more frequent globular vegetations in the arterial chambers of the heart.

As yet, chemical analysis has contented itself with demonstrating the quantitative excess of fibrin in the blood. According to our own researches, however, investigations are urgently requisite which have for their principal aim to determine the qualitative impairment of the fibrin. An augmentation of the fibrin is always coupled with a diminution in the amount of blood-globules, and, as chemists maintain, at the same time with an increase in the proportion of fat present in the blood. This certainly, however, does not apply to every fibrin-crasis.

Fibrin-crases attended with great exsudation, frequently bequeath, as consequent upon the exhaustion of fibrin, a condition of hypinosis [defibrination], and of hydræmia. They eventually prove fatal from this source, if the patient have escaped the deadly influence of paralysis of the organ of localization, or of spontaneous coagulation in important sections of the vascular system; for instance, in the ramifications of the pulmonary artery. The highest grades of dyscrasial fibrin-constitution, finally degenerate into sepsis of the fibrin, and, indirectly, of the entire blood-mass.

The crasis may also terminate in restoration of the normal crasis, through conversion of the morbid excess of fibrin into nitrogenous substances, eliminated with the urine and perspiration. The fibrin thus becomes largely converted into excrementitious matter.

(*a*) SIMPLE [ORGANIZABLE FIBRIN-YIELDING] FIBRIN CRASIS.

It is the attendant upon inflammations with an organizable exsudate,—that is, an exsudate susceptible of textural conversion. It comprises the inflammations of wounds healing by the first intention; many inflammations of glandular organs, and of serous and synovial membranes which terminate, not in purulent liquefaction of their products, but in gradual resorption or in textural conversion of the latter,—or in the case of pneumonia, in obliteration of the pulmonary texture.

The product of these processes—that is, the exsudate-fibrin determined by these processes—answers to the character of fibrin 2. [See "Fibrin."]

The crasis consists in this: namely, that the fibrin, besides increase in quantity, usually manifests, within the blood-vessels, the character of the exsudate-fibrin just adverted to; in other words, those qualities which fibrin acquires in certain processes of inflammation. The tendency of fibrin to coagulate is sometimes aggravated into spontaneous coagulation within the vascular system.

The coagula are white, or yellowish-white, compact, frequently holding inclosed a notable quantity of serum. Under a closer inspection they appear as a glebous, fibro-glebous blastema, here and there delicately fibrillated in wavy curls. Through this are interspersed numerous black-contoured, spherical or fibre-drawn nuclei, along with scattered, dull-granular nuclei, and nucleated cells. All the nucleus-formations are rendered more sharply defined by the influence of acetic acid, the blastema itself becoming turgescent and transparent. [See "Fibrin 2."]

To this category belong not a few of the so-called vegetations or fibrin-coagula within the heart's cavities, not a few coagula in blood-vessels of every calibre down to the capillaries, perhaps also the intra-arterial stratiform coagula, and those endogenous depositions which are the primitive source of phlebolites.

Unless these coagula—produced during life—be, in a state of minute subdivision, taken up again into the blood, they enter into a textural conversion.

The crasis is either a spontaneous, primitive, or else a consecutive one engendered by infection of the blood with a product of a corresponding kind.

The dead subject is marked by great cadaveric rigidity, by firm, deep-red muscles, by tense, dry areolar tissue, and by retarded lividity and decomposition.

(*b*) THE CROUPOUS CRASIS [PIORRY'S HÆMITIS].

The *croupous crasis* occurs under several forms, which at the same time represent various gradations of disease of the fibrin. Amongst them we find, on the one side, the most marked hyperinoses; on the other, a scanty proportion of fibrin, but that deeply affected in quality. This is manifested first by its augmented coagulability, by a greatly increased tendency to deposition in the shape of intra-vascular coagulation [in the capillaries, as capillary phlebitis], and, lastly, by acute processes of exsudation.

Both the intravascular coagula and the exsudates are distinguished by their indisposition to become organized, by their early liquefaction, and very often by their corroding, solvent effect upon the textures. Both are opaque, yellow, or of a greenish yellow, and contain fat. The adhesive property gradually diminishes.

To the latter, namely, the exsudates, must be reckoned some, exhausting by their volume and abundance, others inferior in quantity, but indicating, by their tendency to liquefy and by their reaction upon the textures, the deep impairment of the fibrin.

They are often idiopathic, but more frequently consecutive crases, emerging out of others,—the typhous, the exanthematous, &c. In the former case they are marked hyperinoses; in the latter case they are determined by inflammation, and the infection of the blood with congenial products. They constitute the so-called hæmites of Piorry.

In their processes of exsudation, they evince a preference for the mucous membranes, especially of the respiratory and of the digestive tracts, as also for the serous and synovial membranes.

CROUPOUS CRASIS *a*.

It is characterised by the following relations of the fibrin in its coagulation and exsudation.

The coagula—engendered in the death-agony—are, in the heart, either clod-like, cord-like, more or less compact masses prolonged into the blood-vessels, or, where the energy of the heart's systole has been broken long prior to death, and the mortal struggle protracted, membranaceous, lining the heart's cavities or insinuating themselves in the shape of fangs amongst the trabeculæ. When developed during life, they appear, in the heart, as the liquefying so-called globular vegetations; in wide-calibred blood-vessels, as cylindrical and plugging, or as membranaceous coagula loosely attached to the internal membrane of the blood-vessel; in capillary ranges, as obstructions of the texture varying in circumference. They are either pure fibrin or contain more or fewer blood-globules, incorporated with them during the act of precipitation. In the former case, they are marked by their opacity, by their dull white, yellowish, or yellowish green coloration. In the latter case, they are likewise opaque, but, according to the amount of contained blood-globules, more or less reddened.

A closer inspection shows the coagula to consist of a glebous-like, fibro-glebous blastema, or of a faintly striated, membranous basement,—like the inner, the fenestrate, blood-vessel membrane, bestrewn with point-molecule, with numerous granulated, grayish nuclei or nucleus-like formations, and with similar granulated cells. All the nucleus formations are uninfluenced by acetic acid, except that a slight shrinking takes place, and that they acquire a somewhat sharper outline. Not unfrequently the entire coagulum seems to consist of these nucleus- and cell-formations, along with a proportion of point molecule. [See "Fibrin 3."]

The metamorphosis of these coagula consists for the most part in a tolerably rapid liquefaction of the blastema to a puriform, tenacious fluid holding the aforesaid form-elements in suspension.

The exsudates, reflecting the hyperinotic condition of the blood, are generally very abundant, even to the exhaustion of the fibrin. They are reddened in a degree conformable with the amount of extravasated blood which they have incorporated,—as, for instance, in hepatization of the lung. Or they are of a grayish yellow with a slight shade of green, and opaque. Their metamorphosis consists, possibly with textural transformation of any organizable portion, in disintegration and liquefaction of the blastema to a pus-like, tenacious fluid. [See "croupous exsudate *a*"].

CROUPOUS CRASIS β.

Its characters are nearly those of the former, as regards the outer aspect of the coagula and of the exsudates, only more strongly developed. Thus, the opacity of the coagula and of the exsudate-fibrin is more considerable; their coloration, where they do not include blood-corpuscles, more decidedly of a greenish yellow. Their metamorphosis consists in rapid, puriform liquefaction.

More narrowly scrutinised, the coagula are found to consist of a fine, dense point-molecule, of nucleus- and cell-formations in different degrees of completeness and of assimilation to the pus-cell. These are held together through the instrumentality of an amorphous bond-mass. Engendered during life, they break up—the blastema liquefying—into a tenacious fluid, in which the elements specified are held in suspension; and which, in proportion as the cells predominate, more and more resembles pus. [See "Fibrin 4."]

The exsudates, corresponding in character with the usually hyperinotic condition of the blood, are generally very copious, exhausting, of a yellowish or a greenish-yellow tint, imperfectly adherent to the exsudation-surface, rapidly liquescent to a puriform fluid, and of a form-composition identical with that of the coagula. [See Croupous Exsudate β.]

In both the coagula and the exsudates, the basement or blastema connecting the form-elements has lost the fibrillation so characteristic of coagulating normal fibrin, and even the glebe-like structure.

Certain conditions are common to both varieties of the croupous crasis [α and β]; to the latter, however, they apply in a higher degree.

Both become localized in the shape of exsudatory processes upon the mucous membranes, especially of the respiratory tract; in early youth, in the larynx and trachea; at a later period, in the bronchia; and from the period of puberty to the end of life, in the lungs [as laryngo-tracheal, as bronchial croup, and as croupous pneumonia]. Upon the mucous membrane of the womb, as also upon the great serous sacs, especially the peritoneum, with or without congenerous puerperal metritis, they become located, as puerperal processes; in the synovial sacs, as acute rheumatism; again, as endocarditis and inflammation of blood-vessels; lastly, in the areolar tissue, the pia mater, the spleen.

They also become localized as the metastatic deposits of capillary phlebitis.

The high grade of the internal dyscrasial influence is no doubt the cause why a protracted stasis is not requisite for product-formation; why, therefore, the exsudation takes place very rapidly; and why, notwithstanding the great bulk of the exsudate and the lax and vulnerable nature of the textures, it is not hemorrhagic; in other words, why it is not attended with any extensive laceration of the blood-vessels. There is little doubt that the pneumoniæ stated by Hodgkin to enter at once into yellow and rapidly liquefying hepatization, belong to this class.

By long contact, the deliquescent coagula and exsudates frequently exert a solvent, corrosive power upon the textures. In this manner they determine fresh inflammation, ulcerous loss of substance, phthisis of the organs, secondary phlebitis, pulmonary abscess, phthisis of the peritoneum, of the pleura, and, along with these, of the abdominal and thoracic parietes.

Or again, they become re-absorbed, or else, owing to the changes attendant upon their disintegration, they undergo, together with partial absorption, fatty conversion, usually followed by inspissation and cretefaction.

One further phenomenon here finds its elucidation, namely, *the milky blood*. This has been witnessed in pneumonia and peritonitis, and we have ourselves encountered it in a developed form in pneumonia, and in very intense inflammation of the spleen. Such blood has been found to contain an excessive proportion of fat; it is, however, questionable whether this be the sole cause of the phenomenon. We believe the latter to be due rather to the disintegration of croupous fibrin within the circulation, in other words, to the suspension of the point molecule [the molecular fibrin] in the blood-serum. It is very possible, indeed, that a fatty condition of the blood may contribute to produce the milky appearance; and not improbable that in some cases of a different kind, for example, in the blood of dram drinkers, it may be the sole cause.

The rigor mortis manifests itself in the inverse ratio of the magnitude of the effusion, and the same inverse relation obtains between the latter and the intra-vascular fibrin-coagula. The blood is in part loosely clotted; for the most part fluid; owing to simultaneous loss of serum through exsudation, tenacious; and of a dark cherry-red [defibrination]. It forms, in every variety of organs, dirty-red hypostases; death-patches become rapidly and extensively developed; the liver is dark-coloured. The muscles are lax, the parenchymata collapsed, flabby, lacerable, moist, and where there is no hypostasis, pallid.

CROUPOUS CRASIS γ. APHTHOUS CRASIS.

In the croupous crases hitherto described hyperinosis commonly prevails; in the crasis we are now entering upon, the amount of fibrin in the blood is for the most part scanty. By so much the more significant must be the anomaly in the *quality* of the fibrin and in the general crasis, and we shall presently see this to be really the case, if we extend the idea of this crasis in a natural order beyond that constitution of the blood proper to ordinary aphthæ as affecting the mucous membrane of the mouth.

The exsudates appertaining to the croupous crases before described occasion destruction of the textures only after long-continued contact with them, and in their secondary phase of disintegration. In the aphthous exsudates this effect is produced at once, and as it would seem during the process of exsudation itself.

The exsudates are yellow, greenish-yellow, dingy-gray, opaque, tough products, which, upon superficial expansions, coagulate in a pseudo-membranous form, and, together with the textures, rapidly break up. They are very often remarkable for the exuberant epiphyte-formations [thread-funguses] which take root in them. The disintegration manifests itself in various forms doubtless connected most intimately with the grade and with the modification of the crasis, from simple corrosion with secondary reactive inflammation in the vicinity [aphthæ], down to fusion to a variously discoloured, fetid, ichorous pulp, or to a tough or a friable slough that tears like soft agaric.

With this the crasis has attained the phase of a putrid decomposition, a sepsis, a necrosis of the fibrin and of the general circulating fluid. The blood is discoloured and contains partly tough, partly pap-like, discoloured, ichorous, coagulate fragments—as exemplified in diphtheritis, sloughing tonsils, puerperal putrescence of the womb, septic dysentery, &c. The seat of such processes is above all in the mucous membranes, especially of the alimentary tube, of the urinary bladder, of the female sexual organs and their follicles; in open wounds and sores, in areolar tissue, in common integument. Amongst them may be reckoned thrush [diphtheritis]; certain exsudation-processes upon the mucous membrane of the intestinal canal, especially of the colon [as a form of dysentery], and of the uterus after delivery; corrosive fibrin-exsudates upon the mucous membrane of the urinary bladder; upon external and internal sores; muco-membranous ulcers, especially upon the base of the typhous ulcer; white gangrene of the common integuments [hospital gangrene].

The aphthous crasis is often idiopathic. It is, however, no less frequently consecutive upon other anomalous crases, and upon the typhous, the typhoid, the exanthematous in particular. The blood, reduced in quantity, presents the characters proper to those crases; the few coagula themselves, the characters of excessively croupous fibrin.

The dead body is collapsed, devoid of rigor. The death-patches become speedily discoloured to russet and green. Muscles, flabby, pale. Parenchymata, collapsed, friable. The blood, wasted, dusky red, fluid, with a few villous, broken coagula, marked by opacity, toughness, discoloration, and septic disintegration.

(*c*) THE TUBERCLE-CRASIS.

Although there are, no doubt, tuberculoses purely local, a tuberculosis extending through several organs, or even through one entire organ, is invariably the offspring of a tuberculous dyscrasis.

The latter is for the most part developed out of a *fibrin-crasis*.

In the dead subject, a hyperinosis of the blood may not be ostensible.—Nay! owing to the frequent and voluminous processes of exsudation attendant upon the course of a tuberculosis, a poverty in fibrin is perhaps more likely to reveal itself after death. Excess of fibrin has, however, been demonstrated, in such cases, by the chemical examination of blood withdrawn during life, and it may also be inferred from the extent of the tuberculous deposition. Still the hyperinosis is far from being adequate to account for the disease. It is the qualitative anomalies of the fibrin that must determine its tuberculous nature,—anomalies of quite a peculiar kind, which, as we shall see, may modify every one of the fibrin-crases referred to into the tuberculous.

The gray tubercle answers to the character of the *simple fibrin-crasis* [See "Gray Tubercle"].

The opaque, yellow, lardo-caseous tubercle, marked by its proneness to undergo softening, answers to the character of the *croupous-crasis* [See "Yellow Tubercle"].

We have remarked of the products of the fibrin-crases that they are seldom unalloyed. The same observation applies to the products of the tuberculous-crasis. The products of the one always occur intermingled with elements of the other; and tuberculous products may even include more or less of organizable elements which form into textures in the ordinary way.

Again, the transitions of the individual tubercle-crasis from one to another are obviously brought about step by step. The croupous tubercle appears but rarely as the primitive tubercle. It is generally based upon a pre-existent gray tubercle, and the croupous tubercle appears as an aggravation of the simple form.

In the fibrin-crases a minimum of fibrin suffices for the groundwork of a qualitative impairment, and this in its amplest sense applies to tubercle. The smallest proportion of fibrin present in the blood takes on the taint and becomes expended, up to the point of complete defibrination, in the deposition of tubercle.

Still these said crases by no means serve to throw much light upon the nature of tuberculosis. They must needs involve a peculiar—a tuberculous--modification, the existence of which is indeed proved by the tuberculization of extra-vascular fibrin in hemorrhage, and also of intra-vascular fibrin-coagula. *In this modification must be rooted:*

(*a.*) The surpassing proneness of the fibrin to deposition, so frequently brought about in quite an insensible manner;

(*b.*) The assumption by the deposited fibrin of the particular form of tubercle. The granulation of tuberculous products of inflammation upon serous membranes might indeed be ascribed to a separation of the tuberculous portion, due to its great coagulability. Still the very localization of the tubercle-crasis, in such minute and sharply-defined areæ that little granule-shaped exsudates are the result, constitutes a peculiarity—a remarkable peculiarity, seeing that the same form of separation—the same form of tuberculization—attaches to the fibrin of hemorrhage and to endogenous vascular coagula.

(*c.*) The fact that blastemata resulting from the tuberculous fibrin-crases do not undergo the metamorphoses—proper to the pure fibrin exsudates—of textural formation on the one side, and of rapid purulent liquefaction on the other; but throw off their exsudate-water in the act of firm coagulation, and tarry for a while in this crude state of consolidation. This respite is of various duration, but at all events exceeds both that proper to textural conversion, and that common to disintegration, in corresponding, *purely* fibrinous exsudates.

As the most marked and obvious phenomena connected with tubercle must be specified its high grade of coagulability, and its surpassing proneness to deposition,—to the localization of its crasis. In these are without doubt centred the peculiarities of the fibrin-crases in their tuberculous modifications.

If, in relation to these peculiarities of tuberculous fibrin, we take into account:

(*a.*) The consummate coagulability of arterial fibrin generally;

(*b.*) Its supreme sensitiveness towards heterogeneous substances, such as inflammatory products, whose reception, for example, in arteritis, occasions locally so rapid an obstructing coagulation of the blood-column, as to obviate any infection of the general blood-mass from that point. [See "arteritis," vol. iv.]

(*c.*) The ready deposition of fibrin out of arterial blood, as stratiform coagula upon the inner arterial surface, a disease, in its consummate form, peculiar to arteries.

(*d.*) The very common localization both of the fibrin-crases, whether spontaneous or determined by infection, in the shape of exsudate, or of endogenous coagulation within the capillaries of the arterialising organs [the lungs], and again the still more marked relation of the tuberculous-crasis to these viscera.

(*e.*) On the one side, the pre-eminent development of the pulmonary organ, as predisposing to the fibrin-crases in general, and to their tuberculous modification in particular;

(*f.*) On the other side, the pre-eminent immunity afforded by exquisite venosity and cyanosis, against fibrin-crases, more especially the higher (croupous) grades, and most particularly against the tuberculous-crasis,—taking, we say, all these circumstances duly into account, we are forced on to the momentous conclusion that *arteriality*, that is, *the arterial development, of the fibrin, pre-eminently constitutes the cardinal character of tuberculosis.*

The qualitative impairment of the blood-fibrin here again, as in the fibrin-crasis of a higher grade, serves to explain summarily the fact, that in individuals with blood impoverished in fibrin,—or even generally impoverished,—tubercle continues to become deposited. It has been elsewhere affirmed, and it is worthy of repetition here, that in such cases *every atom of tuberculo-dyscrasial fibrin becomes expended in the formation of tubercle.* This view clears up the seeming inconsistency of affirming tubercle to be rooted in a fibrin-crasis, whilst florid tuberculosis is found to be associated with a deficiency in fibrin. It is the prevailing notion of mere *excess* in fibrin that we would impugn.

The tuberculous-crasis itself may, by various chances, become modified through a combination with other dyscrasial constitutions of the fluids, giving rise, at least in part, to the several known varieties of tubercle.

The tuberculous-crasis is commonly protopathic. Frequently enough, however, it results from other crases. The conversion of typhous, of exanthematous hypinosis to the tuberculo-croupous-crasis is frequent, and of the greatest scientific and practical interest.

The tuberculous-crasis results, through exhaustion of the fibrin, in a defibrinated condition of the blood,—in albuminosis. And, again, the albumen—upon which the wasted fibrin may be supposed to have ingrafted its own morbid character—may take up the work of exsudation in the shape of acute, albuminous tubercle, of lardaceous infiltration of the liver, spleen, and kidneys,—of genuine albuminuria;—hydræmia and anæmia being the final issue.

The corpses of the tuberculous, responding to the long-continued and copious expenditure of protein substances upon tubercle, present general emaciation, with consumption of the fat and of the bone-medulla;—flabbiness, wasting, pallor of the muscles; fatty infiltration of the liver, spleen, and kidneys; and lastly, œdema and dropsy. The blood, with a few scanty fibrinous coagula, is fluid, adhesive, dark-red; or else, with the exception of very inconsiderable soft coagula, it is thin, watery, of a pale-red, resembling water in which flesh has been steeped. Wherever abundant fibrin-coagula are met with, they present the characters proper to the fibrin-crasis, often in conjunction with tubercle-like concretions, which in their elementary composition fully harmonise with the tubercle of exsudation, that is, the tubercle of the textures.

PYÆMIA. PUS-BLOOD.

This crasis, again represents a *local pus-production,* and also a *spontaneous primitive pyæmia* of the entire blood-mass.

In pyæmia it is necessary to distinguish two different grades or stages, in order to bring the various facts into mutual concord, and to avoid contradiction in the characteristic given of this blood disease. Those facts are, on the one side, the coagulation and deposition; on the other side, destruction, of the fibrin. The latter may become developed as a higher grade,—as a consecutive stage, out of the former. The higher grade may, however, set in at once, as a protopathic-crasis, without being preceded by the first or lesser grade.

In the lesser grade the blood-crasis is characterised by quantitative excess in the production of fibrin [hyperinosis], which is at the same time qualitatively impaired. It is marked by a high degree of coagulability and of proneness to separation from the blood-mass,—manifesting itself as croupous, liquefying fibrin. These main features of the crasis illustrate the following appearances.

The fibrin-coagula endogenous to the vascular system, are remarkable for their opacity, for their varying hues of dull white, of yellowish green, of reddish gray [from inclosed blood]. They are soft, and yet tough—their contained serum being of a whey-like turbidness. A closer inspection shows them to consist of a glebous basement, with the rudiments of fibrils, and about these a vast quantity of fine point-molecule. There are, besides these, nuclei, and nucleated cells the nuclei of which appear to resemble in various degrees, up to complete dissilience, pus-nuclei. Sometimes they exhibit little tubercle-like congeries, which consist of collected elements of pus. Having originated during life, they soften, with liquefaction of the said basement, to a fluid which assimilates to pus proportionately to the amount of pus-cells included in the clot.

This crasis, in its most developed grade, possesses the peculiarity of localizing itself in many areæ in rapid succession. Along with highly acute inflammation with purulent effusion upon mucous membranes, upon serous membranes, and in areolar tissue, these areæ appear in every variety of organ and of texture, and are generally distinguished for their small circumference and their sharp definition. They form suddenly in the textures as red obstructions, which almost as speedily deliquesce with sloughing and ulcerous fusion of the involved textures to a yellow or greenish-yellow pus. Wherefore,—as also owing to the lack of evidence of antecedent inflammation,—they have received the name of pus deposits or depôts; of pus metastases. We have already described the attendant anatomical process, and seen that these acts [like others brought about under the fibrin-crases] consist essentially in a spontaneous coagulation of the blood-fibrin in the capillaries, and its immediate liquefaction, with ulcerous corrosion of the blood-vessel membranes and of the contiguous textures; to which process, inflammation with similar products, as the encompassing inflammatory areola, supervenes. Coagula of the same nature form in the great vessels and, in the shape of purulent vegetations, also in the heart.

The blood appears, along with the endogenous fibrin-coagula described, as a tenacious fluid of a russet hue. It is seen spread out in a thin layer, and mingled with little soft particles, which turn out to be aggregates of pus-nuclei and pus-cells, along with blood-corpuscles, in a transparent clot.

The dead body, owing to the voluminous separation of coagula, presents extensive livid patches verging upon russet coloration; hypostases; textural redness of imbibition; lack of rigor; flabby muscles; friable, doughy, collapsed parenchymata.

The higher grades of the pus-crasis consist in destruction of the fibrin; attenuation and discoloration of the blood; septic decomposition of the circulating fluid, of a nature corresponding to the rapid ulcerous and gangrenous up-breaking of textures in the local processes.

The more intense is the aggravation of the crasis, the less do we encounter the aforesaid depôts. It is only in the transition to the higher grades that we meet with them, obviously breaking up, along with the involved textures, to a dingy brown, coffee-ground, or olive-coloured, collapsing, fetid pulp. The same metamorphosis affects both the exsudates and their parent strata. In the *highest grade* extensive passive stases affect the decomposed blood, producing necrosis with dark and hemorrhagic imbibition of the textures.

The dead bodies of persons who have died of pyæmia at this stage, manifest, apart from the external and internal local processes—due, it may be, to an earlier phase—long retention of animal warmth, little and evanescent rigor, flabby and pale muscle, more especially discoloration and lacerability of the heart, and rapid decomposition with extensive, brownish death-patches. The parenchymata are lax, easily torn, serum-drenched, pale, or of a spurious redness in various shades, owing to hyperæmia and imbibition of dissolved blood-pigment. The lungs are the especial seat of hypostatic congestion, with a dark coloration verging upon cherry-red or upon brown. The blood in the heart and vascular trunks contains scanty, colloid-like, cruor-holding, red, and sometimes greenish-red, coagula, and is itself of a cherry red, adhesive, or else attenuate, brownish; having stained the blood-vessel membranes and the endocardium with its colouring matter. Pyæmia is not unfrequently *primitive* [protopathic—or deuteropathic, that is arising out of other anomalous crases—for example, the typhous, the exanthematous]. More commonly, however, it is *consecutive* to the reception of pus into the blood, or else to infection, brought about in the various ways, fully detailed at page 365 of the present volume.

This applies to both grades of pyæmia of which it has been stated that the second also occurs independently, determined through infection of the blood by a foul pus decomposed through stagnation. To this category belongs, amongst others, the infection proceeding from the poisoning of wounds with pus out of the dead body. Darcet brought forth the disease by injecting corrupt pus-plasma, a disease into which, as a purulent sepsis or necrosis of the blood, the minor grades of pyæmia with hyperinosis become spontaneously exalted, probably owing to the pus in the blood becoming oxydized in the respiratory process.

The circumstance that pus secreted bodily into the canal of a blood-vessel, commonly produces a more intense infection than pus-plasma probably received into the blood by resorption, might lead one to conclude that it is to the pus-cell that pyæmia is attributable. Nevertheless, apart from the incomparably larger proportion of pus received into the blood in the former case than in the latter, the occurrence of primitive pyæmia precludes our adjudicating in favour of either the pus-cell or pus-plasma as the causal agent. Far more depends, without any doubt, upon the quality of the pus; and it is certain that a large proportion of bland pus taken up into the circulation proves far less mischievous than an incomparably smaller quantity of purulent ichor. That the pus-cell taken up into the blood, or even that other cell formations of larger size—certain cancer-cells, for example—should, by obstructing the capillaries, give rise mechanically to so-called depôts [metastases] will scarcely be credited at the present day.

Pyæmia generally proves fatal, as purulent poisoning: inconsiderable grades of it, however, are susceptible of cure. This occurs, without doubt, partly through a conversion of the pus, analogous to the metamorphosis of fibrin—partly through elimination of the pus in exsudatory processes, especially upon extensive mucous membranes, like that of the intestinal tract. The elimination of pus through processes of secretion, for instance, its passage through the kidneys, with the concurrent disappearance of pus-deposits, is a phenomenon much talked of. The pus-cell both in the urine and in exsudates is incapable of becoming reabsorbed, and equally so of passing out of the blood-vessels, either into the uriniferous tubules or at any other part. It follows, therefore, that pus-cells, either in the urine or in an exsudate, must be a new creation out of effused plasma.

2. VENOSITY, ALBUMINOSIS.—HYPINOSIS (SIMON.)

This constitution of the blood is characterised by *deficiency in fibrin,* but *preponderance of albumen, and generally speaking, also of blood-globules.* The blood is upon the whole thickish, tenacious, dark-red, and contains, if any, only a few soft, gluey or jelly-like coagula, in which there is much cruor pent up.

It *has a very extensive domain,* comprising a vast number of special crases, which reveal their kindred nature by the general characters of the blood just defined, by the metamorphoses which many of them undergo in common, and by the general sameness of their products; whilst again they differ in some particular attribute of the latter, and by specific relations to particular textures and organs [Localization.]

Their range comprehends the most important and most perilous, acute and chronic diseases; plethora [general hyperæmia], venosity of the lungs, and heart diseases, the acute exanthemata, especially scarlatina and measles, the so-called substantive fevers, *chronic* rheumatism and gout, rickets, typhus, Asiatic cholera, so-called acute tuberculosis, Bright's disease, and lardaceous degeneration of the liver, spleen, or kidneys, mollities ossium, cancer, the crases of acute convulsions, of tetanus, of hydrophobia, diseases of the nerve-centres, chronic mental alienation, hypochondriasis, chronic metal-poisoning, especially with lead, narcotism, finally the crases accompanying atrophy after acute, exhausting diseases, the so-called suffocative death-seizures generally.

To discover the nature of the special crasis in so heterogeneous states, is reserved for the future, and rather for chemistry than for anatomy.

Many acute crases issue in *septic destruction of the albumen* and putrid decomposition of the entire blood-mass. This consummation is especially frequent in the exanthematous and the typhous crases, and in acute convulsions. The acute crases are moreover liable to frequent transformations, especially to the croupous crasis and to pyæmia. There occurs frequently an acidifying of the blood, which localizes itself in miliary eruption and in acute softening of the stomach.

The crasis is sometimes *protopathic,*—habitual, persistent, ingrained in the individual, or acute and evanescent. At other times it is *deuteropathic,* or the sequel to exhausting and especially to defibrinating disease. It is, moreover, a primitive blood-disease, called forth by poisons, by miasma, by contagion, or else it is consecutive to disease of solid parts [for example, organic heart disease], and determined by neurosis.

The products placed under its control [exsudates and new growths] are distinguished by an excess of albumen, by very subordinate coagulability, by lack of disposition to become organized, by persistence at embryonic grades of structural development.

A not unfrequent sequel to extensive exsudation is hydræmia, or, it may be, tarlike inspissation of the blood with anæmia. The former becomes developed without any notable serous effusion, the water being otherwise disposed of in the morbid process. The anæmia is commonly due to a shattered condition of the nervous system.

Several of these acute crases have a decided relation to the mucous membranes, and especially to their follicular apparatus, to the lymphatic glandular system, to the common integuments, to the spleen. The dead body presents, especially in the acute crases, dark coloration of the common integument; rapidly developed, extensive, and very dark death-patches; early decomposition; a very marked but for the most part evanescent rigor, and a lax, doughy condition of the parenchymata. Hyperæmiæ and stases arising in the different organs, not unfrequently become exalted into hemorrhage. In the tarlike inspissation of the blood, the corpse is in a high degree emaciated or rather shrunken, dry,—the common integument, of a lead-colour, or livid.

Let us endeavour to submit the more important of these crases either singly, or where the distinctions are not very marked, more collectively, to an anatomical muster.

(*a*) PLETHORA.

It is characterised by *excess of blood, by a preponderance of the blood-globules over the fibrin, by a deep red, tenacious blood.* It involves the direct manifestations of venosity in the inverse ratio of the amount of blood which the organism is capable of arterializing. It occurs under two opposite and contrasting relations. First, in conjunction with florid nutrition of the textures, fullness of muscle, and especially ample areolar tissue and fat formation. Secondly, as a very marked phenomenon in union with general emaciation,—wasting of the solids [so-called nervous tabes]. Under the latter circumstances, it is observable both in very delicate children, during the first months of their life, and in insane adults [in hypochondriasis, melancholia, &c.].

In the dead body, the general overloading of the vascular system, and occasionally surpassing hyperæmia of various organs, especially of the lungs or of the brain, or of the liver and entire portal system are manifest. According to the degree of intensity of the crasis, all the soft parts are more or less deeply coloured. In the emaciated, the common integuments exhibit vast patches of a purple, or of a bluish leaden hue.

Plethora predisposes to congestion, to hemorrhage, to blennorhoid, albuminous, and serous exsudations of greater or less moment in proportion to their amount and to the importance of the organs concerned. In corpulent, square-built [apoplectic] individuals hyperæmiæ of the lungs are frequent. In these the plethora often of itself, but more commonly through acute serous effusion into the bronchia and lung-cells, proves speedily fatal. Moreover, the plethora occasions dilatation of the heart, with subsequent, progressive augmentation of its substance [hypertrophy].

(*b*) THE TYPHUS CRASIS.

It compasses the entire nature of typhous disease, and is at the root of all its phenomena, whether of substantive change or of functional disturbance.

The typhus crasis is marked by the destruction—the diminution—of the fibrin, and the comparative preponderance of the blood-globules. The typhous blood is in various degrees fluid, and of a deep purple colour. It forms, if any, but scanty, loose, soft, and humid, deliquescent coagula, reddened by the imbibition of pigment-holding plasma.

The corpses of typhous individuals are remarkable for the deep, dingy, bluish gray coloration of the common integument, for the deep purple of the death-spots, for the dark russet hue and the rigidity of the muscles, and for the dryness of the areolar tissue. The serous membranes, and especially the peritoneum are of a dull gray, lack-lustre, and occasionally suffused with a tenacious humour. All the textures in contact with blood appear discoloured from imbibed hæmatin, of a peculiar shade, verging from violet colour upon brown.

In the next place, the multifarious local hyperæmiæ have to be noticed. They are due to the paralysing influence of the blood upon determinate ranges of the nervous system, either at the periphery or at the centres. Foremost amongst them are local hyperæmiæ of the mucous membranes, of the lungs, of the brain, and its membranes, of the spinal cord, of the common integuments. They often display the attributes of so-called hypostasis. Upon mucous membranes they frequently degenerate into hemorrhages, which occur also, although far more rarely, in parenchymata, for example, in the brain.

The typhus-crasis manifests a very marked relation to mucous membranes, especially to the lymphatic glands and to the spleen. In middle Europe it is the mucous membrane of the intestine and especially of the ileum, rarely the bronchial mucous membrane with the lungs and the bronchial glands; in the North, it is rather the last mentioned, namely, the respiratory tract; in the south, [in pest-typhus] it is the peripheral lymphatic gland system, in which the crasis becomes localized. In the form of a typhous inflammation it determines, in the follicular apparatus of the ileum and in the mesenteric glands a peculiar marrow-like product which, in intense cases, closely resembles medullary carcinoma.

The very variable consistency of the typhus-substance points to variations in the typhus-crasis itself; to different degrees of plasticity in the typhous blood plasma.

Pus-formation, we have to observe, *is alien to the genuine typhous process* whether general or local. Wherever it does occur, it is founded in a degeneration or change in the typhus-crasis, of which we have to say a few words. No other crasis offers such manifold interest in reference to *degeneration* or *conversion.* Not alone are there several conversions of the kind, but they are remarkable for an impress the exact reverse of the original typhus. The recognition of these phases and their interpretation as degenerations or transformations, are not only of the greatest scientific interest, but also of the most obvious practical utility. These changes resolve themselves into the following. They are more or less demonstrable in the sanguineous fluid, as also more or less proclaimed in corresponding local processes.

1. *Conversion to the croupous crasis.*
2. *Conversion to pyæmia.*
3. *Degeneration to acute softening* [acidification of the blood].
4. *Degeneration to gangrene* [Sepsis,—necrosis of the blood].

1. *Conversion to the croupous crasis.* A fibrin of a constitution characteristic of the croupous crases forms in the blood. Perishable coagula [vegetations, plugging clots, so-called capillary phlebitoids] originate in the heart, in the greater vessels, in the capillaries; but, above all, exsudative processes, upon mucous membranes. Those croupous inflammations of the mucous membrane lining the tracheal canal, the œsophagus, the stomach and intestines, the female sexual organs, as also croupous pneumonia, all belong to this type. Again, similar processes upon serous membranes, the yellow, fibrinous crumbling products by which the typhous infiltrations of the mesenteric and Peyerian glands are modified.

The exsudates are wont to exert a solvent influence upon their parent strata, deep corrosions of the mucous membranes beneath the exsudates, more especially at the glottis and epiglottis, being not at all uncommon.

This conversion takes place at various periods of the typhous process—even at a very early stage. It is deserving of notice that a vast number of cases in point happen at the commencement of epidemics of cholera, a disease in whose typhoid [so-called reaction-] stage croupous inflamations are so frequent. [See "Exanthematous crasis."]

The conversion of the typhus-crasis *to the tuberculous*—the *tuberculo-croupous*—*crasis* belongs to the same class. Its localization generally attaches to the lungs in the shape of lobular—not unfrequently of a comprehensive lobar pneumonia;—pneumonic tubercle-infiltration.

2. A *second conversion* of the typhus-crasis, kindred with the preceding one, is that to *pyæmia* and *local pus-production*. It occurs, for the most part, at a later period than the one just referred to, often complicating the last stage of the local typhus-process upon the mucous membrane of the ileum, and protracting itself into a *sequela* to the typhus. Examples hereof are the pus-deposits in the typhous patches upon the mucous membrane of the ileum, and in the infiltrated mesenteric glands; the pus-producing areolar-tissue inflammations; the purulent exsudates upon serous tunics; the circumscript, suppurating coagulations in the capillary system of the lungs, the spleen, the kidneys; the boil-like obstructions of the capillaries in the substance of the mucous membranes and of the outer skin.

3. *Degeneration* to the *acute softening process*, that is, to a crasis in which the latter is founded, and which is localized in softening of the stomach, &c. We believe this process to be a peculiar one, quite distinct from putrid decomposition and its characteristic, gangrenous sloughings. That which concerns us here occurs as black, or Indian ink-coloured softenings or meltings of the textures in an acid fluid, especially in the cœcal sac of the stomach, on the left side of the œsophagus, in the lungs, upon the mucous membrane of the cœcum, and in the urinary bladder.

We believe *this process to be derived from the blood in the capillaries of the parts referred to, and to be due to an acidification of the blood-mass,—to the presence of a free acid in the blood:*

(*a.*) It is developed out of a hyperæmia and stasis in the implicated organs, and in point of fact, out of the blood engaged in the stasis, which experiences the first effect of the liberated acid upon itself, in the shape of inspissation and coagulation to a black, pitch-like, friable mass, destructive of the walls of the vessels and of other contiguous textures.

(*b.*) The reaction of the structures softened is invariably acid.

(*c.*) Our view seems to derive support from the determination of the blood under these circumstances to the cœcal sac of of the stomach, which, with the spleen, appears to us to per-form the office of a de-acidifying apparatus to the blood-mass, for the immediate secretion of the gastric juice and in behalf of the hepatic function.

(*d.*) A very frequent appearance associated with the impending softening, is that of a miliary eruption with acid reaction of the contents of the vesicles.

4. *Degeneration to sepsis; putrid-crasis.* Primary gangrene of the solids. It occurs either very early, or only as a sequel to typhus.

The blood and the dead body exhibit the peculiar changes to be described in another place.

Where the putrid character is early developed, the faint impression in its localization, especially with respect to plasticity of its products is remarkable. The Peyerian gland-groups are turgid with sero-albuminous infiltration, lax, and, together with the mucous membrane of the ileum, generally ecchymosed.

As the local manifestation of this degenerate state, sloughing takes place in parts exposed to hypostasis and pressure, for instance, in the sacral region, at the trochanters, &c. This is not all however: hyperæmiæ and stases become developed even in parts beyond the range of hypostasis, leading incontinently to mortification of the textures,—for example, noma of the cheeks, sphacelus of the external sexual organs in the female.

All these degenerations may become localized in the typhus-ulcers, leading, as will be seen in the account given [See vol ii.] of the local typhus-process in the intestinal membrane, to a destruction overstepping the limits of the textures, and frequently to perforation of the intestine.

Other sequelæ of the typhus-crasis are protracted albuminuria [Bright's disease], anæmia [with wasting], hydræmia [Œdema, Dropsy].

(*c*) THE EXANTHEMATOUS CRASIS.

Its domain, viewed from the anatomical side, is a very extensive one. However we might wish to limit this crasis to scarlatina and measles, a number of acute blood diseases naturally cluster around them, become localized upon the greater mucous membranes, and not unfrequently, especially in epidemics, produce exanthemata presenting more or less analogy with genuine measles, or pure scarlatina. Amongst them are some which determine a more or less plastic, albumen-loaded, coagulating or colloid-like, thinly purulent, almost serous product upon mucous membranes; exsudatory processes which bring the muco-membranous texture into a state of dissolution; Asiatic cholera; numerous puerperal affections; acute diarrhœa, especially in children; nay, even exanthematous typhus; in fine, many substantive [exanthematous] fevers without exanthema. At the uttermost limits of this domain are placed blood-diseases, associated with tonic spasm and convulsions,—with affections of the nervous centres. Last of all, acute tuberculosis. In the present section, however, we shall only speak of the exanthematous-crasis, and of those nearest allied to it, leaving the remainder to be discussed in separate chapters. The crasis in question is the most distinctly marked in scarlatina; and it is here that we have the best opportunities for studying it in the dead body. In degree, the crasis of measles is perhaps nearly the same, as are also the blood-diseases already stated to follow next in the scale.

Upon the whole, *the exanthematous crasis has the greatest affinity to the typhous. Only the blood is still more fluid, whilst the violet tint present in intense typhus is wanting. The blood verges more upon purple,—upon cherry-red.*

The dead body manifests a certain degree of turgor. There is a lack of that tenseness of the muscles, and of the common integument, as also of the dingy-gray coloration of the latter. The skin is indeed rather white, although with extensive, very saturated death-patches. The serous membranes very often exhibit a viscid, ropy, colourless covering. Local hyperæmiæ, partly of hypostatic nature, and imbibition of the textures with blood-pigment, are observable.

Along with these differential points, and apart from the resemblance in the anatomical characters of the blood, *special analogies come forth between the typhous and the exanthematous crasis.* Such as:

(*a.*) *The relation of the exanthematous crasis to the mucous membranes, and also to the lymphatic glands.* It is expressed in the well-known catarrhal, erythematous, and other affections of the respiratory and gastro-enteric tracts of mucous membrane; — in the well-known enlargements of peripherous lymphatic glands accompanying the course of the exanthemata. What is, however, particularly characteristic, is the development of the follicle apparatus of the ileum and of the mesenteric glands in scarlatina, and in the entire series of analogous blood-diseases.

(*b*.) The identical conversions of the crasis observed in typhus. The most frequent are the conversion to the croupous-crasis, including the tuberculo-croupous, with corresponding local products, and the often early degeneration to putrid decomposition.

Among the sequelæ there is one proper to typhus, and also a very frequent consequence of scarlatina,—namely, protracted albuminuria.

Another consequence of the exanthematous processes, is intense inspissation of the blood, with marked hyperæmiæ and stases.

As far as its crasial source is concerned, variola does not seem to constitute an exception. It is, however, essentially obnoxious to a speedy transition into the croupous-crasis and into pyæmia, the latter often outrunning its normal term. In common with the exanthematous-crasis generally, it is liable to degenerate early into putrid decomposition, which, anticipating the croupous and the pyæmic phases, precludes the formation of any products due to these modifications of the exanthema, and causes the pocks to degenerate into the so-called putrid.

Amongst the diseases following closely in the wake of the more prominent exanthemata, the under-mentioned are peculiarly deserving of notice, namely:

A large proportion of puerperal fevers—especially when bearing an epidemic impress. The characters presented by the dead body, and the anatomical relations of the blood answer to the exanthematous crasis; to which may be added, the presence of the exsudatory processes above specified upon the uterine mucous membrane, the character of exsudates detected upon the serous membranes, especially the peritoneum, and finally, the frequency of concurrent exanthema, in the shape of erythema and of scarlatina. These puerperal processes are marked by their tendency either to become converted into the croupous crasis, or else to degenerate into putrid decomposition. Nor is acute softening of the stomach a rare coincident phenomenon.

A crasis appertaining to this class often becomes localized, as one of the *exsudatory processes adverted to, upon the intestinal mucous membrane*, as in many instances of *acute diarrhœa* and of *dysentery*. These often prove fatal through paralysis of the intestine — through exhaustion. A very remarkable and momentous phenomenon, however, is a resulting thickening of the blood to a dark red liquid, of a tarry appearance, and of the consistency of treacle. It proves fatal under the symptoms of anæmia in vital organs [lungs, brain], of rigor [tonic spasm] of muscular organs, or else through local hyperæmiæ [for example, of the brain]. Eruptive phenomena in the progress of the dysenteries are not at all uncommon.

Conversions of the crasis to the croupous and to protracted pyæmia are frequent.

Next in the series is the *cholera-process*. It is a more or less rapidly developed hypinosis, with the characters of the exanthematous. Its localization extends over the entire intestinal tract as an exhausting exsudatory process, multifarious in its products, and either proving rapidly fatal under acute inspissation of the blood and the aforesaid contingent phenomena, or else passing over into a so-called stage of reaction.

In the former case, the dead subject presents a dingy, blue-gray coloration of the common integument—a puckered state of the latter and of the areolar-tissue, with herb-like dryness and rigidity, and with dark coloration of the muscles. The blood, if we except certain stases in different ranges of the vascular system, especially in the blood-vessels of the membranes of the brain, is found accumulated in the vascular trunks, and in the heart, as a dark, tar-like mass, without fibrin-coagulum. The lungs are for the most part dry, inflated, of a deep red; the serous membranes—more especially the peritoneum—moistened with an abundant viscid coating. The intestinal canal, is the seat of an extensive, and equally rapid and intense process of exsudation, and presents the general lineaments of paralysis. Surcharged as it is with fluid, it is nevertheless collapsed, soft, and flabby-membraned, pallid, rarely presenting any intussusception. It contains, in varied measure, a serous fluid, rendered turbid, whey- or rice-water-like, whitish or yellowish-white, by the débris of epithelium and minute particles of protein substances, or else slightly reddened by the intermingling of blood. The mucous membrane is denuded of its epithelium, and bare; or the coagulable portion of the exsudation may adhere to it in the shape of a loose bran-like covering, or of membrane-like formations. Its texture is bloated, and is for the most part readily scraped off, as a reddish-white pulp; its follicles, especially at the ileum, distended by exsudate to the bigness of millet or hemp-seeds. The spleen is shrivelled, the urinary bladder empty. In the ganglia of the sympathetic we detect little hemorrhagic spots, as big as poppy or millet-seeds.

In the so-called stage of reaction, the crasis reverts to the normal, or else the hypinosis changes under expansion of the blood into a typhoid disease. The latter is remarkable for a secondary localization upon the mucous membrane of the intestines, in the shape of repeated processes of exsudation, and also for exanthematous processes simulating measles, scarlatina, pemphigus or erysipelas, upon the common integument. During the thus protracted course of this hypinosis, it is very usual for a fibrino-croupous-crasis to develop itself, and for the mucous membrane of the intestine, of the stomach, of the œsophagus, of the trachea, to display croupous exsudation,—the lungs, croupous pneumonia.

This hypinosis is moreover convertible to pyæmia, to acute softening, and also liable to putrid decomposition.

(*d*) HYPINOSIS IN DISEASES OF THE NERVES.

The similarity of the crasis in this class of diseases with the typhous and exanthematous-crasis is very striking. It even partakes, in common with these, of a proneness to localize itself upon the intestinal mucous membrane in the follicular apparatus of the ileum,—in a turgescence [product formation] of the Peyerian and solitary gland capsules.

To this category belong diseases with obvious anatomical disturbance in the nervous centres, and again diseases in which such disturbance is either wanting or subordinate and consecutive. Such are meningitis, acute hydrocephalus, apoplexy, and the like; and again, acute tonic spasm and convulsions, tetanus, trismus, puerperal convulsions, protracted epileptic convulsions, &c.; lastly, hydrophobia.

In the latter diseases, more especially, in which up to the present day, no anatomical disturbance is demonstrable, the question arises as to whether the nervous system be substantially impaired at all,—whether the anomaly in the crasis be not the primary cause of the nervous phenomena.

It appears to us that, although the most accurate examination may be inadequate to prove any palpable anatomical disturbance, a primitive affection of the nervous system must nevertheless exist, and be that which determines the [secondary] anomaly of the crasis.

This crasis not unfrequently degenerates into putrid decomposition. It often becomes converted into pyæmia, and not unfrequently issues in acute softening.

In fine, those rapidly destructive liquefactions of the blood may be here classified, which, under the name of asphyxia furnish forth the majority of *instances of sudden death*, commonly through hyperæmia of the lungs with acutely developed pulmonary œdema.

(*e*) THE DRUNKARD'S DYSCRASIS.

That dyscrasial condition induced by the abuse of alcoholic drinks, and especially of gin, occurs under two forms, differing in the course which they run—in other words, there is *a chronic* and also *an acute drunkard's dyscrasis.*

The first, namely, the *chronic crasis,* manifests itself as plethora, with a remarkably dark coloration, a thickness and a simultaneous fattiness of the blood. This occasions, and at the same time accounts for, the condition in which we find the solids.

The pigmented appearance of the skin, the excessive, and, at the same time, anomalous fat-formation,—and blennorrhœæ are all characteristic of the crasis. The corpses of inveterate dram-drinkers present very marked appearances. The skin is tinged of a dingy brown, and this is coupled with the fact, that not unfrequently parts naturally rich in pigment—the scrotum, for example, become deprived of it. At the same time the skin is of a soft, unctuous feel, like that of the negro, and its epidermis layer is thin. Subjacent to the skin, and also in the mesentery and the omenta is deposited fat, in an excess if not absolute, at least relative to the state in which we find the muscles, and possessing a peculiar character not unlike that of mutton suet. Together with this, the muscle-flesh appears to have lost in volume, and to have become pallid. The fat formation steals into the muscles in the shape of fatty conversion. The liver has undergone fatty degeneration. Even in the bones the fat formation has gained ground at the expense of the bony texture.

All the mucous membranes, but especially the bronchial and intestinal, are affected with chronic thickening and with blennorrhœa, the chronic gastric catarrh [gastric irritation] being particularly marked. A similar state of hypertrophy presents itself in the habitually congested cerebral membranes, in the form of dullness, thickening, chronic œdema.

The brain is affected with an atrophy like that met with in the aged, with or without considerable dilatation of, and serous effusion into the ventricles.

The blood appears dark coloured, grumous, defibrinated, viscid-unctuous to the touch, often intermingled with fat in large quantity, as fat-drops. In rare instances the disease occasions a chyle-like opacity of the plasma—milky blood.

The chronic drunkard's crasis often undergoes conversion to *fibrin-crases* of various kinds. Amongst these are inflammations with fibrin-products, even tubercle. Pneumonia is a very frequent and very fatal disease with drunkards—pneumonia running an acute course, and possessing an eminently croupous character. Chronic hepatitis, determining organizable products, and leading eventually to liver-cirrhosis [granulation] is a very common termination.

In drunkards tuberculosis runs an eminently chronic course. The deposition of tubercle is for the most part inconsiderable; the granulations are generally of a dingy, or a greenish-gray, and do not soften as such. The yellow tubercle and resulting phthisis are more rare.

The natural issue of the drunkard's crasis is in eventual hydræmia—in dropsy—which assumes a local form, especially that of ascites, the more speedily, the earlier heart-disease or liver-cirrhosis becomes established.

The acute crasis has a marked resemblance with the exanthematous, and with the crasis in nervous affections. The liquefaction with discoloration of the blood and, as a consequence thereof, the tendency to transudation of blood-serum, are for the most part more developed. The fat is wont to separate from the blood in the form of largish drops.

The corpses present extensive, very saturated death-patches; evanescent rigor of the dark red muscles; congestion of the cerebral membranes, and still more of the lungs, especially as hypostasis; scattered patches of stasis in the intestinal mucous membrane, &c. The parenchymata are lax; those affected with hyperæmia, imbibed with a coloured blood-serum. In the cavities of the serous membranes—more especially of the pleura—are dingy-red, serous [spurious hemorrhagic] effusions. The corpses emit a peculiar sweetish smell, and pass rapidly into decomposition under gas development.

The inflammatory stases developed during the progress of this crasis are, for the most part hypostatic pneumoniæ. They determine a product dark-coloured from adherent hæmatin, lax, soft, incompetent to hepatize the lung-texture.

This crasis never becomes developed in aged persons broken down by repeated attacks of delirium tremens, but invariably in drunkards in the early years of manhood, who are endowed with a powerful muscular system. It runs a very rapid course, leading, in a very few days to decomposition.

We are aware of no instance of this crasis passing into the fibrinous crasis, or into pyæmia. On the other hand, it often becomes exalted into decomposition, and not unfrequently issues in softening of the stomach.

It is not improbable that this crasis is, in the majority of cases, due to injury sustained by the brain during a violent or protracted fit of intoxication, and that it ought rather to have found a place in the preceding chapter. It is not in aged drunkards, with an atrophied brain, that it occurs, but in younger individuals with a brain of normal development, keenly sentient of congestion, and of an alcoholized condition of the blood.

(*f*) THE CRASIS OF ACUTE TUBERCULOSIS.

This crasis has the greatest resemblance with the exanthematous, and the disease assimilates so closely in its manifestations during life to typhus [intestinal typhus], as only to be distinguishable from the latter by the absence of abdominal symptoms, the more marked phenomena pointing to exsudation in the membranes of the brain and upon the linings of its ventricles.

The product of this crasis is a tubercle presenting many peculiarities. It is a scattered corpuscle, mostly smaller than a millet-grain, and no bigger than a poppy-seed, or even a pin's point. It is now of glassy transparency, vesicle-like; now grayish, semi-opaque, soft, gluey;—then, again, verging upon whitish, or yellowish-white, and opaque. With it there always exsudes a grayish, more or less albumen-sated, semi-gelatinous serosity, infiltrating—drenching—the involved textures. The deposition of these products always affects an organ in wide extension. Generally speaking, indeed, several organs are implicated at once, more especially the lungs, the cerebral pia mater, the spleen, the liver, and the serous membranes. The tubercle is always deposited in great numbers, and is equably disseminated throughout an organ, as the examination of an involved lung or spleen shows at a glance.

The tubercle is sometimes primitive, but more commonly successive to a pre-existent fibrin-tuberculosis. Looking at the physical properties of this tubercle, apart from any chemical analysis, and connecting it with the crasis of which it is the product, we are constrained to set it down as an albuminous formation.

With reference to the crasis itself, which, as we have said, is marked by a hypinosis closely resembling the exanthematous, it may be asked [and the question is replete with interest]: is acute tuberculosis primitive, or is it but a consecutive state of defibrination of the blood, brought about through the antecedent out-throwing of a large proportion of fibrin, in the shape of tubercle?

Or is it one of the so common tuberculoses connected with hydrocephalic effusion of the internal membranes of the brain, that is, consecutive hypinosis, determined by disease of the brain?

Seeing that acute tuberculosis occurs under conditions which preclude either of the contingencies here referred to, not a doubt can remain as to its protopathic character.

A further question is: how, in this hypinotic crasis, is the tubercle brought about?

[For a reply to this question the reader is, to avoid entire repetition, referred to the section on "Albuminous Tubercle," in the present volume, p. 326.]

Acute tuberculosis probably always proves fatal.

Occasionally, owing to very extensive deposition, the crasis approximates to hydræmia. It does not pass into putrid decomposition, nor is it converted into pyæmia. On the other hand, acute softening of the stomach is a frequent follower in its train.

The corpses present, generally speaking, the same phenomena as in typhus; namely, pale skin, extensive, deep-coloured patches of lividity, tense, dark-coloured muscles, hypostatic hyperæmiæ. The parenchymata, especially those which have been the seat of tubercle-deposition, are turgescent, and drenched with sero-albuminous fluid.

(*g*) CANCER-DYSCRASIS.

A crasis the existence of which is shown from the exclusive relation stated, in the general section on tubercle, to exist between cancer and tuberculosis.

To demonstrate a cancer dyscrasis from anatomico-clinical data is one of the most difficult tasks. The basis of such a demonstration is the immediate character of the blood, the peculiarity of the cancer-formation, and of other exsudates brought about in various ways under the crasial influence; and, lastly, their relation to new-growths proper to other known crases.

The blood itself affords evidence of a *hypinosis*. This is, however, not of itself alone cognizable as a specific cancer hypinosis. To prove this the presence of cancer-formations is indispensable, and even these must needs give evidence of their *general* import, either by redundant growth or by multiplication; in short they must in some way betoken a direct relation with a dyscrasial state of the blood.

Further evidence respecting albuminosis is afforded by the presence of albuminuria, of lardaceous infiltration of the liver, spleen, kidneys; more particularly, however, by inflammatory products, as albuminous, white, emulsion-like, in part slowly solidifying, ulcerating, or cancer-forming exsudates; lastly, by the exclusion of concurrent fibrinous products, and especially of fibrinous tubercle.

The abundant fat-formation not unfrequently co-existent with carcinoma may be cited in proof of the participation of fat in the cancerous albumen-crasis. This is exemplified in osteo-porosis from the excessive formation of bone-medulla; in the fatty contents of the cancers, and of albumino-cancerous exsudates; in the deposition of fat as cholesteatoma, as gall-stone, &c.

The cancerous hypinosis manifests its impress in various grades. It is intense in cases of voluminous, exuberant cancers; in very widely spread cancer-production, whether spontaneous, or called forth by the extirpation of bulky carcinomata; but most especially in cancer of acute growth, and of the medullary character. The blood often contains, in nucleus- and cell-formations, the elements of cancer. In chronic vegetation, and especially in pure fibrin-cancer, the hypinosis is often less marked; whilst, in cancer of local import, it may be altogether wanting.

The cancer-crasis is either primitive or consecutive, that is, developed out of a *hitherto local* cancer. It is either acute or [more often] chronic.

The acute crasis is in rare instances protopathic; more commonly, however, it is developed out of the chronic, especially after the extirpation of extensive cancers. It localizes now in the more vigorous growth of a cancer already in existence, now in the simultaneous or in the successive, hasty production of new cancers [of the medullary form] in the most various organs and textures, conducing thus to rapid wasting of the blood, and proving fatal within a term not exceeding that of the most acute crases known.

In its chronic development it terminates in marasm of the blood, in hydræmia, in anæmia, the more readily, in proportion as the seat of the cancer [in the stomach, for example] is calculated to interfere with the work of nutrition, or in proportion to the loss of blood by hemorrhage.

Under such conditions the cancer-crasis may wear itself out, and the cancer-tumours participate in the waste and decline of the entire organism. This explains the circumstance that in a venosity verging upon hydræmia and depending upon central organic impediments to the circulation, cancers hardly ever occur.

The cancerous hypinosis is, as we have already pointed out, absent in local carcinoma. The concurrent crasis may be the normal, or some anomalous one not of a cancerous nature.

The *fibrin-crases*, however, accompanying, or at least coincident with cancer, are of great interest, not alone, as running counter to our theory of the nature of cancer, but more particularly because, if correctly seen and comprehended, they afford the best means of demonstrating the specific character of the cancer-crasis.

1. In the first place, it is conceivable that fibrin-crases may become developed along with local cancer. They are, no doubt, sometimes primitive, localizing themselves in the local cancers, as inflammations; sometimes consecutive, that is, brought about in the cancer itself by mechanical or medicamental influences. The fibrin-crasis concurrent with local cancer may even be of a tuberculous character, and lead to tuberculous deposition.

2. A fibrin-crasis may, however, become developed even conjointly with cancer of general import, that is, out of cancerous hypinosis or albuminosis. The cancer-crasis is coordinate with other, similar [hypinotic] crases, out of which we have seen that fibrin-crases, more especially those of a croupous character, may emerge. They may arise either directly out of the hypinosis, as a conversion of the latter, or else through the instrumentality of an inflammation with cancero-dyscrasial blood, in which a development of fibrin takes place.

The cancerous fibrinosis, in whichever way brought about, localizes itself in inflammations of the serous tunics, in carcinomato-fibrinous hepatisations of the lung, as also in spontaneous coagulations within the vascular system, including the capillaries [cancero-capillary phlebitis]. Both these and the exsudates are distinguished for their opacity, their whiteness [changed by contained blood-globules to grayish-red or red], their soft, lax consistency, their albuminous contents, their medullary characters. They are sometimes fundamental to cancer-formation,—the most acute and most extensive cancer-formation,—both intravascular and extravascular. At other times they liquefy to a white, cream-like, lardo-glutinous ichor. They contain the rudimental elements of cancer in redundant quantity.

In the description just given, a *peculiar constitution of the fibrin* under the conditions both of its organizability and of its liquefying tendency, is undeniable. It is essentially proper to cancer, and affords incontestable proof of the specific constitution of the albumen in cancerous hypinosis. Where a fibrin crasis develops itself, whether in the totality of the blood or in a local process [inflammation], this *peculiarity* of constitution is, without doubt, transferred from the albumen to the fibrin. A proof, this, how intimately it clings to both substances; a proof of the existence of a cancerous fibrin-crasis; and at the same time an indication of the sense in which the balance between cancer-crasis and fibrin-crasis is to be understood.

This *cancerous fibrinosis, in fine, is the parent of a peculiar tubercle* of cancero-fibrinous character, which corresponds well with cancer-crasis, and more particularly with such of its highest grades as have attained the point of fibrinosis; a tubercle, moreover, which, as we have seen at page 313, answers in all respects to cancerous fibrin.

3. HYDRÆMIA; ANÆMIA.

(*a*) THE SEROUS CRASIS; HYDRÆMIA.

Fibrin, albumen, blood-globules, are here all diminished in quantity; *the amount of water increased.* The blood is attenuate, watery, pale in various degrees to the point of water in which flesh has been steeped, wanting in tenacity. It contains very inconsiderable, loose, soft, curd-like coagula holding much serum, which, by pressure, is reducible to a few drops.

The water transudes through the parietes of the vessels in dependent parts, or in such as, owing to mechanical influences, are particularly obnoxious to hyperæmia, drenches the textures in the form of œdema, especially the areolar tissue, even to the medullary system of the bones, and forms, in serous cavities, dropsical effusion. It may transude pure, or may contain a certain proportion of albumen and even of fibrin, which latter [as so called spurious fibrin] determines in the textures a soft curd-like coagulation of the dropsical fluid; and in the cavities separates in the shape of soft curd-like flakes. Inflammatory products are marked by the large amount of their serous contents, and by their poverty in plastic materials.

The dropsical crasis occasions defective nutrition, with pallor of the textures, relaxation of the contractile fibre; in the dead body, the development of pale death-patches.

It becomes mortal through insufficiency of nutritive matter in the blood; but for the most part proves fatal at an earlier stage, through local œdema of the textures, and dropsy of the great serous cavities.

Not every dropsy is, however, the result of hydræmia. We allude to those local and general dropsies brought about by mechanical impediments to the circulation in the veins, in the heart and great vascular trunks, and in the lungs.

The serous crasis is sometimes idiopathic, produced by climatic relations, by peculiar alimentation, by anomalies in the chylo-poietic system, by repeated, exhausting hemorrhages, &c. Nay, it may be even congenital and constitutional. The condition of the blood in hæmorrhophilis, seems to be essentially that of hydræmia. In most instances, however, it is secondary, developed as a sequela to some other crasis, for example, as a consequence of the habitual outpouring of albumen, the separation of fibrin in large aneurysms, the deposition of fibrin and albumen in inflammation-products, in tubercle, in cancer, in albuminuria. Or else it ensues upon a specific, chronic or acute blood-consuming dyscrasis, upon metallic poisoning, typhus, and the like.

(*b*) ANÆMIA.

Deficiency of blood, in various degrees, by no means offers any distinctive crasial characters, if we except the hydræmia—the excess of water—into which every persistent anæmia eventually resolves itself. The anæmia or oligæmia, is brought about in various ways; for, whatever be the crasial constitution of the blood, it is liable to an accidental reduction of its mass.

It is, most frequently of all, a consequence of loss of blood through the various kinds of hemorrhage; next to that, of insufficient alimentation, of excessive bodily and mental labour, and of the continuous loss of fluids; of the inordinate production and increase of new growths, even of redundant fat formation, especially in children; of disease of the nerve-centres, especially of the brain, such as hypertrophy, heterologous growths, concussion. Or it is the sequel and issue of intense typhus-crasis; of chronic metallic poisoning, &c. It accompanies all general atrophy, both in old age and in earlier life.

Moreover, oligæmia is not unfrequently a congenital, constitutional state, and affects by preference the female sex. It involves a corresponding defective development of the calibre of the arteries, with smallness of the heart, and with a generally stunted growth of the animal frame. The female sexual organs seem more especially crippled in their development. It was stated that blood of every admixture may suffer an accidental reduction of its mass through hemorrhage, without becoming alienated from its original crasis. In like manner, anæmia is probably never purely such; that is to say, never brought about by the equable reduction of each of its constituent parts, but at the same time invariably a dyscrasial condition. How inextinguishably the dyscrasis clings to croupous blood, even after the most copious blood-lettings; how, in the highest grades of blood-deficiency, the tuberculous constitution attaches to the smallest remnant of fibrin, we have already seen.

The most striking picture of anæmia is furnished in the dead bodies of persons who have died of hemorrhage. Collapse and pallor are the outward signs reflected from within. The death-marks, if there be any, are very pale. There is considerable rigidity of muscle, firmly contracted heart, presenting the aspect of concentrical hypertrophy, bloodlessness, both of the endocardial cavities and of the vascular trunks, especially the arteries. In corpulent individuals with a white skin, the common integument is of a waxy paleness. In profound dyscrasial anæmiæ [the consequence of typhous or metallic poison] the dead subject retains, together with the pallor, the characteristic cachectic hue.

4. DECOMPOSITION. PUTRID, SEPTIC CRASIS. SEPSIS OF THE BLOOD.

We have repeatedly had to refer to a *decomposition*, a *putrid decomposition* [*sepsis*] *of the blood,* as a consecutive crasis resulting from the degeneration of another crasis.

The conditions we are here concerned with vary, as the anatomical results show, with the causal influences at work, as also with the differences due to pre-existent crases.

Generally speaking, these conditions manifest themselves in decomposition, in dissolution, in necrosis, in *a death* of the blood, and they comprise the commonly called scorbutic, the chronic and acute, putrid states of the circulating fluid. A very broad line of demarcation, coming under anatomical notice, and which separates the states referred to into *two* series is that, *in the one case, the sepsis has the character of a fibrin-crasis, in the other, that of a deficiency of fibrin.* Accordingly, if we except the two common features of thinness and discoloration of the blood generally, a comprehensive view of all these states is not feasible.

Regarded from a clinico-anatomical point of view, the various conditions of septic crasis occur in the following forms:

1. *The purest and most simple forms of sepsis are:*

(*a.*) A decomposition or necrosis of the blood brought about without any cognizable agent of fermentation is that due to a shattered state of the nervous system and of its function, proving fatal with lesser or greater rapidity [sometimes in a very few moments], according to the measure and amount of the shock. To this category are to be referred decompositions of the blood consequent upon concussion and severe injuries, concussion from a fall, from the extensive laceration or the crushing of soft parts or of bones; upon extensive amputations; upon the continuous, exhausting activity of the muscles in violent convulsions, of whatever kind; upon electrical shocks received [lightning]; upon mental emotion of an overwhelming nature. A very striking exemplification offers in the decomposition of the blood not unfrequently called forth by a difficult and exhausting act of parturition involving palsy of the womb; cases which often prove fatal after a very few days, or even hours.

In all these cases the blood is found attenuate, in colour comparable to a raspberry jelly, or of a dingy red, facile of imbibition, expanded in volume, often engaged in gas-development, frothy. The blood-corpuscles are swollen up, the serum being deeply reddened by hæmatin withdrawn from them. Coagula, if present at all, appear as very inconsiderable, soft, curd-like fibrin-clots. The frequent large peritoneal exsudates, occurring more especially in puerperal decomposition, are dingy-red, dull, thin, sometimes rather viscid fluids. The dead bodies present but little and evanescent rigor, much inflation, extensive and deep lividity. The internal organs, the heart's muscle, the parenchymata are lax, flabby; the blood-vessel coats and the endocardium, discoloured from imbibition; those parenchymata whose blood-vessels are most injected are more or less discoloured by imbibed serum. The blood is always largely accumulated in particular sections of the vascular system, be it in the nerve-centres, in extensive patches of the mucous membrane of the stomach and intestines, in the sexual organs of women, but most particularly in the lungs, as hypostasis.

The corpses pass into putridity, under the phenomena of gas-development in the blood-vessels, emphysema in the textures, copious transudation of a dirty-red serum into the serous cavities, and spontaneous vesication of the epidermis.

(*b.*) Those decompositions consequent upon faulty diet [true scurvy], the reception of corrupt matter, of miasmata, of animal poisons into the blood, &c.

The dead bodies present, generally, the characters already specified. Owing, however, to the expansion of the decomposed blood, transudations of blood-dyed serum in the shape of ecchymoses of the textures, and actual hemorrhages are especially apt to take place.

2. *A second form of decomposition* is that so frequently attendant upon hypinotic crases, and which we have before described as an exaltation or degeneration of the typhous, of the exanthematous, of the cholera-, of the drunkard's dyscrasis. Its relation to the fundamental crasis may be regarded as a varying one. Here we may observe:

(*a.*) The putrid decomposition, impelled by a special external agent, becomes complicated with the hypinotic crasis, a septic venom being superadded to the exanthematous contagion or the typhous miasma. There being essential putrid decompositions independent of those other agents, and the symptoms of septic poisoning often occurring very early during the progress of the other diseases cited, such a relation is placed beyond all doubt.

(*b.*) The sepsis is a dissolution of the blood in the hypinotic crasis, resulting from the profound injury inflicted by this crasis upon the nervous system. This implies, either a very intense hypinosis, [a very intense miasma or contagion], or else a very susceptible nervous system. Thus it may happen that typhus and exanthemata pass into putrid decomposition even in epidemics by no means of a malignant type.

(*c*) Or, in fine, it is possible that a hypinosis occasioned by miasma or contagion, may, of itself and without the mediation of the nervous system, become degraded into a putrid crasis, simply through putrid conversion of the received miasma. It is in this sense, more particularly, that the exaltation or the degeneration of a primitive crasis to the putrid is to be understood. The blood and the corpse present the same appearances as in the first form; the marks of decomposition and putrefaction being, however, if possible, still more clearly defined. The different hyperæmiæ are also more distinctly expressed—such hyperæmiæ as are proper to the original hypinoses. They occupy, frequently in the shape of ecchymoses and hemorrhage of the textures into which they have degenerated, those organs or parts of organs in which the original hypinosis had localized itself; for example, the intestinal mucous membrane, in putrid typhus; the common integument and the great tract of the respiratory and intestinal mucous membranes, in exanthematous processes. Not unfrequently, the septic crasis of this form localizes itself in deep-coloured, absolute stases, especially in peripherous organs, where, without a trace of organizable products, they terminate in necrosis of the blood and of the textures, with conversion thereof to a soft, humid, dingy, deep-red mass,—a gangrene-slough.

A stringent differential diagnosis from the blood itself is, however, not-feasible in all the cases of the first and second forms. It is only to be deduced from the anatomical disturbance of the solids generally, and, in cases of the second form, in particular, from the products of the original hypinosis,—typhus, exanthema, &c. For instance, where the intestinal mucous membrane reveals the marks and residua of a typhous process, the sepsis will have arisen out of the typhus-crasis. Where obvious anatomical disturbance does not exist,—for example, in the case of convulsions,—or where the products of a hypinosis, owing to the early supervention of sepsis, are inconsiderable or only faintly indicated, and where, lastly, clinical records are wanting, the diagnosis must needs rest upon probabilities alone.

3. A third form, differing from the two former, is that of a sepsis of the blood resulting from the fibrin-crases, especially the aphthous, and from pyæmia. It manifests itself, in the first instance, as a putrid decomposition, as a necrosis of the fibrin, which forthwith possesses itself of the entire blood-mass. It has been already discussed under croupous crasis γ, and under pyæmia, as aphthous and purulent sepsis.

INDEPENDENT ANOMALIES OF THE BLOOD-CORPUSCLES.

An anomalous relation of the blood-corpuscles, founded in dyscrasial conditions of the plasma, occurs under several forms, some of which have been already adverted to, especially those of turgesence, pallor, or preternaturally deep coloration; of augmented or diminished adhesiveness; and the like. As an independent anomaly, their diminished number in genuine chlorosis is alone recognized.

Other crases deserving of notice are:

The hemorrhagic crasis [hæmorrophilis] specified in the chapter on "Hemorrhage."

A crasis which determines the deposition or stratiform coagulation of a protein substance upon the inner coat of blood-vessels cannot, in our opinion, be pretermitted. The little that we have to advance upon this subject, however, we shall reserve for the chapter on "diseases of the arteries," in vol. iii.

The retention of urea in the blood occasions and sustains a hypinotic crasis; and in certain cases, as in acute nephritis and acute albuminuria, tends to induce a complete decomposition of the blood.

A biliary dyscrasis is produced in two different ways: first, through diseases of the liver and gall-ducts,—inflammation of the liver, obstruction of the latter through retention in the blood of the elements of the bile; and secondly, through endosmosis of bile (resorption) into the blood-vessels.

The croupous-crasis and pyæmia often give rise, without demonstrable liver affection, to the elements of the bile being set at large in the blood.

In fine, there is a spontaneous biliary crasis which, running a very acute course under the most intense typhoid symptoms, and under colliquation of the parenchyma of the liver, proves fatal through decomposition of the blood. So intense and violent is the conversion to bile in the blood, that even in the portal circulation, previous to its entrance into the liver, the blood has the look of blood impaired by artificial contact with bile. It is a dingy-brown or yellow-red, tenacious, ichorous-looking fluid intermingled with whitish fat-streaks and jelly-like particles of fibrin. The bile secreted in the liver is so saturated, and at the same time so excessive in quantity, as to utterly dissolve the parenchyma of that organ,—that is, the hepatic cells—determining a state of collapse and softening, which, in its appropriate place, we shall treat of as "acute yellow atrophy of the liver."

With regard to the constitution of the blood in gout, syphilis, chronic skin eruptions, and many other diseases, although in each a particular anomaly does no doubt exist, it has not as yet been given to morbid anatomy to substantiate its nature.

END OF VOL. I.

EXPLANATION OF THE PLATES.

PLATES I AND II.

Figs. 1 and 4 represent proliferous cyst-formations from the cortical substance of the kidney, as a sequel to Bright's disease. The two figures, 1 and 4, illustrate well Rokitansky's history of proliferous cyst-development, and at the same time what he understands by the often-occurring expression, "alveolar type or arrangement."

In fig. 1 we have the cyst in all its phases. *a* is a simple cyst, arising out of the expansion of the elementary granule, first into the nucleus, from this into the cell, and progressively into the cyst. But it has remained barren, and contains only a diaphanous, viscid serum within a simple cyst-membrane. *b* represents a parent cyst, the *early* history of which accords with that of the barren cyst; within it, however, new granules have formed, and gradually become developed into vesicles or cysts containing other nuclei, until the parent cyst has become replete with them, and, from being spherical, they are rendered polyhedrical by mutual compression. In an adjoining parent cyst, many of the filial cysts have remained barren, others contain nuclei in the act of splitting. *c*, *c*, *c*, *c*, represent another form of development of the parent cyst. Here, again, the parent cyst has gone through the same phases, from the elementary granule upwards. But, as the cell dilates into the cyst, a granule forms centrally to the latter and expands into a filial cyst, centrally to which a third granule opens out in the same manner; and so on. These intra-cystic cysts in their dilatation ultimately close upon the parent cyst, forming secondary, tertiary and ulterior layers, to which an external, fibrous layer is generally added out of the surrounding blastema. Or this fibrous coat accrues in the *alveolar* shape. Fig. 1 affords several examples of this. It is, however, better seen in

Fig. 4.—*a* is the fibrous sheath in progress of development out of *d*, the elongated and caudate nuclei coursing around the parent cyst or aggregation of parent cysts. They eventually break up into the requisite fibres. *e* is to represent the point-molecule, within an amorphous blastema, out of which the nuclei (*b*) form. They are at first spherical, afterwards elongated, and ultimately broken into fibrillation. *This* constitutes what the author designates as the "alveolar type or arrangement." It is, however, still better defined in

Fig. 2, which represents cyst-formation in a medullary carcinoma. From the carcinomatous frame-work a bulb-like excrescence is thrown out, within the extremity of which a parent cyst forms and becomes replete with filial cysts, each containing a central nucleus. This parent cyst surrounds itself with a broad marginal area of blastema, within which elongated, caudate nuclei course round the cyst in several tolerably regular circles or series—the rudiments of a dense fibrous envelope. Such is the "alveolar type," which applies to the fibrous fabric of follicle walls as well as to those of cyst-formations. [See "Cyst and Alveolus."]

Fig. 3 represents a transverse section of a colloid cancer. *a* is an older portion of densely fibrillated fibro-membranous structure. *c* is a transverse section of more recent fibro-membranous stroma; *b*, a transverse section of the colloid warp which intertwines with the said fibro-membranous stroma. [See p. 289.]

Fig 8 represents the multilocular, fibro-membranous stroma of colloid cancer deprived of its colloid contents. [See p. 289.]

Figs. 5, 6, and 7 represent so many stages of the development of medullary carcinoma. They are severally described in the same order in which they are here numbered, at pp. 288 and 289.

Figs. 1, 2, and 4 are magnified by 90 diameters, the five remaining figures by 400 diameters.

Several of the figures here given are embodied from Rokitansky's 'Essays,' in Mr. Paget's admirable 'Lectures on Surgical Pathology,' vol. ii.

Figs. 1, 2, and 4 are derived from Rokitansky's Essay on 'Cyst and Alveolus,' read before the Imperial Academy of Sciences, at Vienna, in 1849; figs. 3 and 8 from his Essay on 'Colloid Cancer,' published in 1852; figs. 5, 6, and 7 from a thesis of his on 'Cancer-stromata,' also published in 1852.

PLATE. I.

Fig. 1.

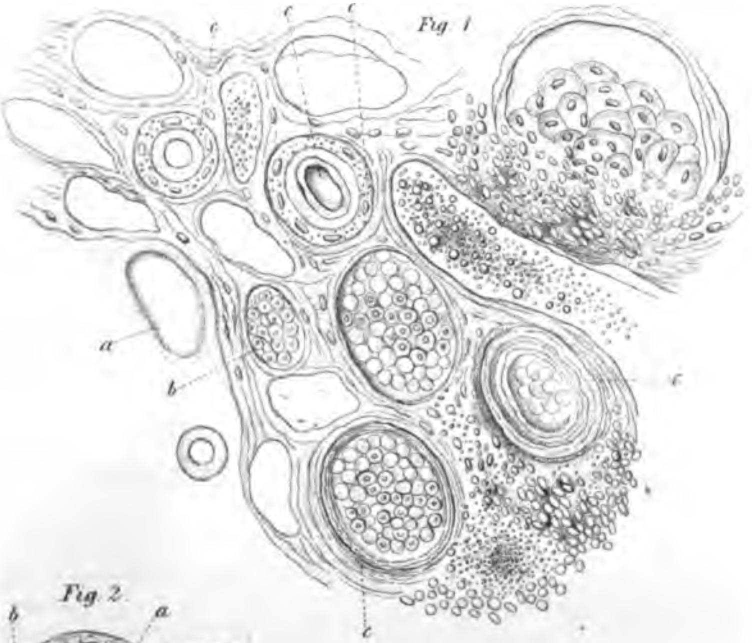

Fig. 2.

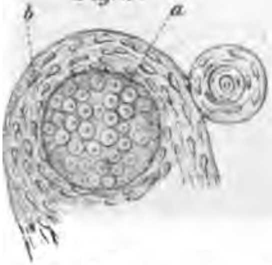

Fig. 3.

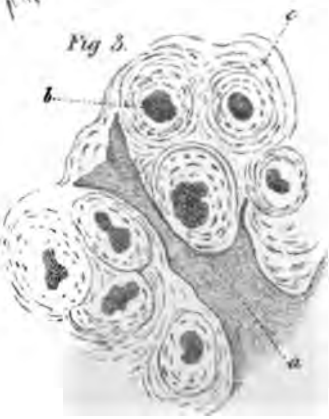

Fig. 4.

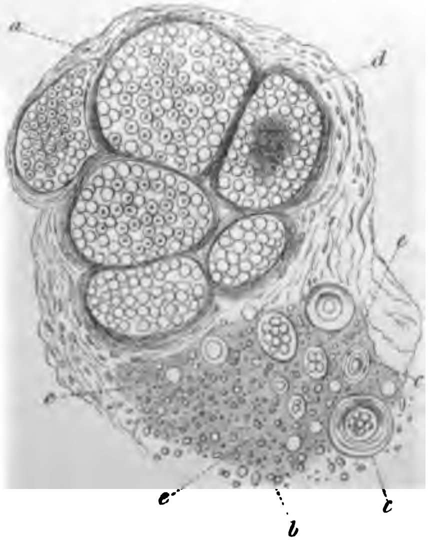

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PLATE. II.

Fig 5

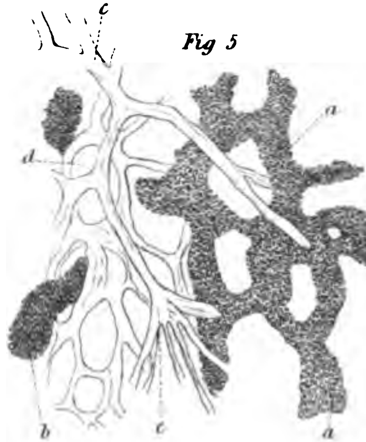


Fig. 6.

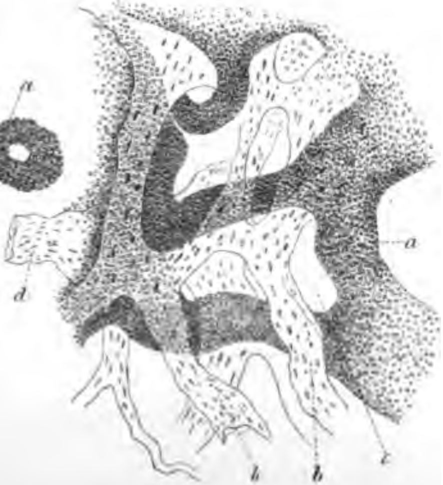


Fig. 7.

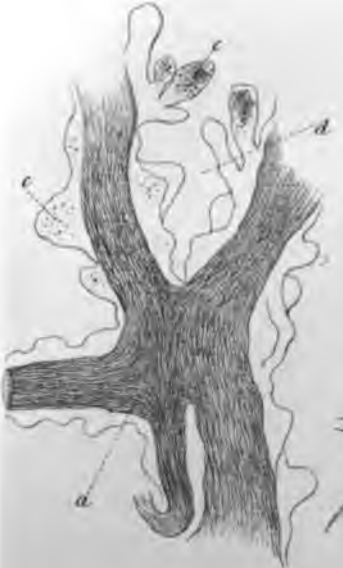
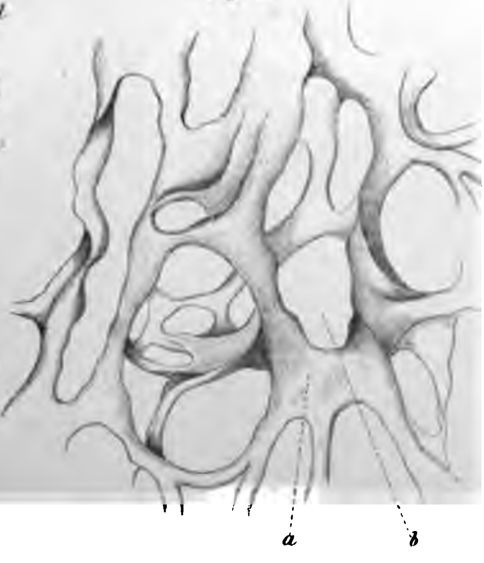


Fig. 8.



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ERRATA.

Vol. I, p. 312, line 13 from below, *for* "with a relatively abdominal," *read* "a relatively small abdominal."
Vol. I, p. 346, line 7 from above, *for* "ascarides," *read* "acarides."

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